CLINICAL AND ANGIOGRAPHIC PROFILE OF PREMATURE CORONARY HEART DISEASE

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CERTIFICATE

This is to certify that this Dissertation titled **"Clinical and Angiographic Profile of Premature Coronary Heart Disease"** is a bonafide work done by **Dr. K.RANGANATHAN,** Post Graduate Student (2009-2012) in the Department of Cardiology, P S G Institute of Medical Sciences & Research, Coimbatore under the direct guidance and supervision and in partial fulfilment of the regulations laid down by The Tamilnadu Dr. M.G.R. Medical University, Chennai for DM Branch II, Cardiology Degree examination.

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DECLARATION

I, solemnly declare that this dissertation titled "**CLINICAL AND ANGIOGRAPHIC PROFILE OF PREMATURE CORONARY HEART DISEASE**" is a bonafide work done by me in the Department of Cardiology, P S G Institute of Medical Sciences & Research under the guidance and supervision of my Professor **Dr. J. S. BHUVANESWARAN, M.D**. **D.M.,** Professor & Head of the Department, Department of Cardiology, P S G Institute of Medical Sciences, Coimbatore – 641 004.

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INTRODUCTION

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Even in the modern era of advanced medical, surgical and critical care interventions, acute myocardial infarction (AMI) remains a disease that is associated with considerable morbidity and mortality. In recent years, its prevalence seems to have been on the increase whereas the mean age of coronary artery disease (CAD) has decreased. Although coronary artery disease (CAD) primarily occurs in patients over the age of 40, many younger men and women can still be affected. Most studies have used an age cut-off of 40 to 45 years to define "young" patients with CAD and AMI.[1-5]. The prevalence of CAD in younger subjects is difficult to establish accurately since it is frequently a silent process and subclinical. An autopsy study of 760 young (age 15 to 34 years) victims of accidents, suicides, or homicides[6] showed advanced coronary atheromata in 2 percent of men and no women. An advanced lesion was present in 20 and 8 percent of men and women aged 30 to 34, respectively, while 19 and 8 percent, respectively, had a $\geq$ 40 percent stenosis of the left anterior descending artery.

There are also limited data on the frequency of AMI in younger subjects. In the Framingham Heart Study, the incidence of AMI over a 10-year follow-up was 12.9/1000 in men 30 to 34 years old and 5.2/1000 in women 35 to 44 years old[7]. The incidence of AMI was eight to nine times greater in men and women aged 55 to 64 years. In few other studies, 4 to 10 percent of patients with AMI were $\leq$ 40 or 45 years of age [8-10]. In two series of patients with CAD at $\leq$ 40 years of age, women comprised only 5.6 and 11.4 % of patients [8-11]. Although CAD is an uncommon entity in young patients, it constitutes an important problem for the patient and the treating physician because of the devastating effect of this disease on the more active lifestyle of young patients. The disease carries a significant morbidity, psychological effects, and financial constraints for the person and the family.

A number of studies have shown significant differences in the risk factor profile and coronary angiographic patterns between young and older patients with acute ST elevation myocardial infarction (STEMI) [12-16]. Traditional risk factors of CAD are prevalent in young patients with acute STEMI but with a different pattern compared to their older counterparts [14, 17, 18]. These differences may cause different treatment strategies and outcome among these patients.

The epidemiology, clinical spectrum of myocardial infarction is significantly different in developing countries compared to the western world. These differences can be attributable to multiple factors including increasing average life span, tobacco use, decreased physical activity, unhealthy diet and psychosocial factors. CAD occurs in Asian Indians 5–10 years earlier than in other populations around the world. The mean age for first presentation of acute myocardial infarction in Indians is 53 years [19-22]. Prevention of these deaths in young people should become a national health priority. A strategy involving prevention of CAD long before its onset will be more cost-effective than providing interventions at a stage when the disease is well established. In India, mortality attributable to CVD is expected to rise by 103% in men and by 90% in women from 1985 to 2015 [23].

While the literature from developed countries is abundant in data highlighting various aspects of myocardial infarction in young patients, only a few studies have been published in India. How far the changes in the epidemiology of MI have occurred in India is not clearly known. There is a need to collect the data from our own community, compare it with the world literature and analyze if the changing epidemiology of MI in young patients is also seen in our country. This study was undertaken with these specific objectives to analyze the clinical and angiographic variables in young Indian patients.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

History of Coronary Artery Disease

Symptoms related to angina pectoris and myocardial infarction were even found in ancient Egyptian papyruses dated approximately 3500 years back. It was in the late 16th and 17thcenturies that a breakthrough was made in the process of understanding this lethal disease. The first study of heart in the world, perceiving relation between loss of consciousness and arrhythmia with an account of ischemia and myocardial infarction was put forth as a text in `Practica Medicinalis' by Bishop Thomas of Wroclaw (1297-1378)[24].

In the 18th century, better knowledge of coronary vascular anatomy lead to the real discovery of the ischemic heart disease and myocardial infarction by a physician who announced his observation in 1768. He was an English physician, William Heberden (1710-1801). It was Heberden who was associated with the discovery and for a long time even in the 19th century, the disease was called as "Heberden's disease"[24, 25].

After Heberden's clinical description of angina, it took almost a century for pathologists to focus their attention on coronary arteries and describe thrombotic occlusions in addition to "ossification". In 1879, the pathologist Ludwig Hekben concluded that myocardial infarction is caused by coronary thrombosis secondary to sclerotic changes in the coronaries[26]. In 1910 two Russian clinicians described five patients with the clinical picture of acute MI[27].Two years later, Sir James. B. Herrick emphasized total bed rest as the treatment for this condition[28] and by 1919 had used electrocardiography to diagnose it[29].

Two seminal developments in the 1960s radically changed our understanding and management of acute myocardial infarction. The National Heart, Lung, and Blood Institute (NHLBI) established the Framingham Heart Study in 1948 with the goal of understanding

how heart disease developed by studying the lifestyles of the residents of Framingham, Massachusetts USA[30]. The first description of their findings, "Factors of Risk in the Development of Coronary Heart Disease," indicated that elevations in blood pressure and cholesterol levels were associated with an increased incidence of ischemic heart disease and acute myocardial infarction. The study also showed a high frequency of MI among women, which often occurred later in life than it did in men. The institution by the NHLBI of national programs to educate clinicians and the public about the importance of controlling these risk factors have contributed to dramatic improvements in age-adjusted cardiac death rates[31].

With the identification of these coronary risk factors and others that followed, the veil that masked the underlying mechanisms in angina and myocardial infarction was lifted, and the concept that coronary heart disease and its complications could be prevented was introduced. Increasingly large multicenter clinical trials subsequently showed that both primary and secondary prevention was possible when steps were taken to lower blood pressure and serum total cholesterol. Fortunately, drugs to reduce these risk factors safely became available as a result of a series of productive collaborations between industry and academic medicine.

Coronary Care Units

Until 1961, patients with acute MI, if fortunate enough to survive until they reached a hospital, were placed in beds located throughout the hospital and far enough away from nurses' stations that their rest would not be disturbed. Patients were commonly found dead in their beds, presumably from arrhythmia. Indeed, the risk of death occurring in the hospital was approximately 30%. The development of the coronary care unit[32], which provided continuous monitoring of the electrocardiogram, closed-chest cardiac resuscitation, and

external defibrillation reduced in-hospital mortality by half among patients admitted with acute myocardial infarction.

Physiology, Cardiac Catheterization, Angioplasty and surgery

The publication of *De Motu Cordis* in 1628, William Harvey's description of the circulation and the function of the heart[33], set the stage for the physiological era several centuries later. The19th-century French physiologist Claude Bernard catheterized animals and measured the pressures in the great vessels and cardiac chambers[34].This experiment led to the first human cardiac catheterization, performed by Werner Forssman on himself in 1929[35], which in turn led to the exploration of cardiac hemodynamics by Andre Frederic Cournand and Dickinson WW. Richards[36]. All three of these investigators were awarded the Nobel Prize in Physiology or Medicine in 1956. Cardiac catheterization paved the way for the development of coronary arteriography in 1958[37].When combined with ventriculography, it allowed clinicians to elucidate the natural history of coronary artery disease. Coronary arteriography and left ventriculography became the standard diagnostic tool for defining pump function and vessel anatomy and provided the foundation for surgical treatment by means of coronary revascularization. The development and refinement of the technique of open-heart surgery required close collaborations among surgeons, cardiologists, anaesthesiologists, haematologists and engineers[38]. The field of invasive cardiology soon emerged, built on the pioneering work of Dotter and Judkins, although Andreas Gruntzig is considered the father of percutaneous interventional cardiology. The initial technique of balloon angioplasty was followed by the insertion of bare metal stents, and today, drug-eluting stents are used to prevent coronary restenosis[39].

Modern Therapy

By the 1970s, in-hospital mortality from acute MI was approximately 15% and in the first year after hospital discharge, roughly 10% of patients died from left ventricular failure (LVF) associated with large infarctions. Studies in laboratory animals suggested that infarct size could be reduced by rectifying the imbalance between myocardial oxygen supply and demand[40]. In 1976, cardiologists opened acutely occluded coronary arteries by intracoronary infusion of the fibrinolytic agent streptokinase[41]. The Italian Group for the Study of Streptokinase in Myocardial Infarction (GISSI) trial, one of the first cardiac "mega-trials" involving more than 10,000 patients, showed that intravenous streptokinase reduced early mortality in patients with acute MI[42]. The Second International Study of Infarct Survival (ISIS-2) showed that the addition of aspirin (an antiplatelet drug) led to further reductions in mortality[43]. Coronary angioplasty and stenting[44], together with newer, more potent platelet inhibitors (P2Y12 and glycoprotein IIb/IIIa platelet–receptor blockers), further reduced in hospital mortality to about 7%. The efficacy of these treatments, including ventricular defibrillation, depends on the duration of symptoms and early arrival to the hospital[45].

Since the 1970s, randomized, controlled clinical trials became the paradigm for the advancement of clinical cardiovascular therapeutics. The Survival and Ventricular Enlargement (SAVE) trial showed that long-term administration of angiotensin-converting–enzyme inhibitors (ACEI) reduced mortality among patients with left ventricular dysfunction after infarction[46]. The use of beta-adrenergic blockers and aldosterone blockers in these patients further reduced mortality. Despite these notable advances, life-threatening heart failure still occurs late in patients with extensive ventricular scarring as a consequence of large infarcts. Implantable defibrillators[47], cardiac resynchronization therapy with pacemakers[48], and left ventricular assist devices[49] have improved the prognosis for such patients. Cardiomyocytes from patients with severe heart failure have been found to be

deficient in sarcoplasmic reticulumCa2+ ATPase (SERCA2a). In a pilot study, an adeno associated virus has been used to deliver the gene for SERCA2a by intracoronary infusion, with seemingly beneficial results[50].

Unstable Angina and Non ST Segment Elevation Myocardial Infarction

In the late 1930s, alert clinicians called attention to what we now refer to as unstable angina and non ST segment elevation acute coronary syndrome. Patients with this disorder have severe anginal pain, usually at rest, often with biochemical evidence of myonecrosis and severe, multivessel, obstructive coronary artery disease. These patients now outnumber those with ST-segment elevation myocardial infarction. Patients with non ST segment elevation acute coronary syndrome have improvement with prompt coronary revascularization and require platelet inhibition with aspirin and a platelet $P2Y_{12}$-receptor antagonist, together with an anticoagulant (low-molecular-weight heparin). Their course after hospital discharge is improved by an intensive reduction in low-density lipoprotein (LDL) cholesterol levels[51] and administration of an anticoagulant[52]

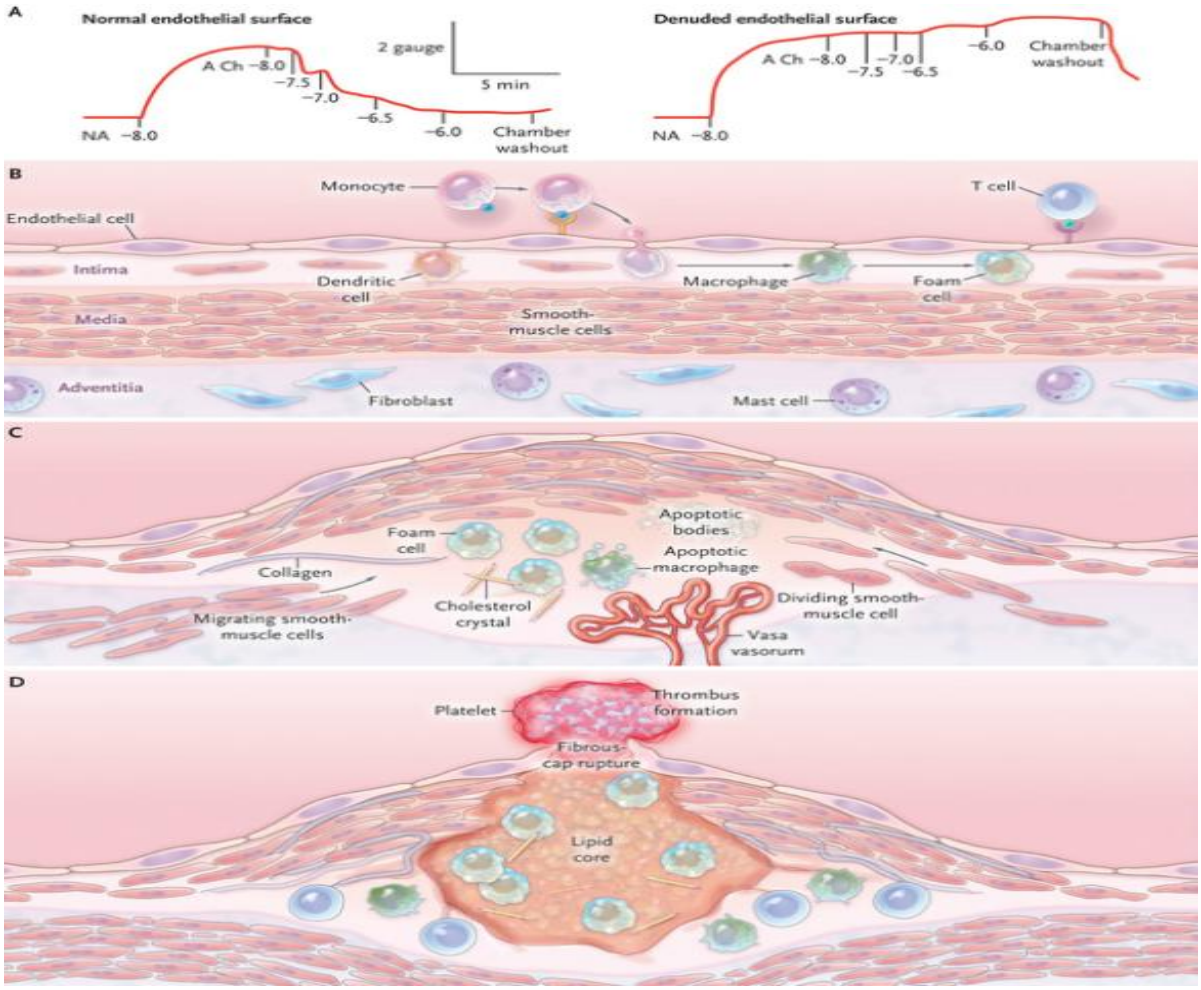
Coronary Atherosclerosis

The ability to access vascular and cardiac tissue rapidly led to the development of animal models of vascular disease, as well as clinical studies in humans. Two lines of investigation in the 1970s and 1980s forged the field of vascular biology: the observations that thrombotic occlusion of a ruptured or eroded atherosclerotic plaque led to acute myocardial infarction [53] and that nitric oxide was a physiological dilator of blood vessels, a discovery for which Furchgott, Ignarro, and Murad received the 1998 Nobel Prize in Physiology or Medicine[54-57]. This pioneering work transformed our understanding of the cellular interactions in both normal and diseased blood vessels and influenced the direction of subsequent research. Investigators shifted their attention from animal preparations of intact

vessels to molecular and cellular regulation and, ultimately, to the genes that encode the growth factors, enzymes, other proteins and RNAs responsible for the development of normal or diseased vessels.

On the basis of these and other studies, we now understand that atherosclerosis is a chronic inflammation of arteries, which develops over decades in response to the biologic effects of risk factors. Atherogenesis begins as a qualitative change to intact endothelial cells when subjected to oxidative, hemodynamic, or biochemical stimuli (from smoking, hypertension, dyslipidemia) and inflammatory factors, they change their permeability to promote the entry and retention of blood-borne monocytes and cholesterol-containing LDL particles. Inflammation and biochemical modifications ensue, causing endothelial and smooth-muscle cells to proliferate, produce extracellular matrix molecules, and form a fibrous cap over the developing atheromatous plaque. Plaques lead to clinical symptoms by producing flow-limiting stenoses (causing stable angina) or by provoking thrombi that interrupt blood flow on either a temporary basis (causing unstable angina) or a permanent one (causing myocardial infarction). Physical disruption (rupture) of the plaque exposes procoagulant material within the core of the plaque to coagulation proteins and platelets, triggering thrombosis[58].

Evidence of the causative role of LDL cholesterol in atherosclerosis is threefold: first, genetic mutations that impair receptor-mediated removal of LDL cholesterol from plasma cause fulminant atherosclerosis; second, animals with low LDL-cholesterol levels have no atherosclerosis, whereas increasing these levels experimentally leads to disease; and third, human populations with low LDL-cholesterol levels have minimal atherosclerosis, and the



process increases in proportion to the level of LDL cholesterol in the blood[59,60]. The LDL-Receptor Pathway and Treatment with LDL Cholesterol–Lowering Drugs, 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins), discovered by Akira Endo[63], were developed. Brown and Goldstein's discovery of the LDL-receptor pathway[61], for which they were awarded the 1985 Nobel Prize in Physiology or Medicine, provided a genetic cause for myocardial infarction in persons with familial hypercholesterolemia and introduced three general concepts to cell biology: receptor-mediated endocytosis, receptor recycling, and feedback regulation of receptors. This last concept is the mechanism by which statins selectively lower LDL-cholesterol levels in plasma, reducing the risk of myocardial infarction and prolonging life, as shown in multiple, definitive clinical trials[62,64].

However, statin therapy does not eliminate cardiovascular risk[65,66]. Levels of high-density lipoprotein (HDL) cholesterol correlate inversely with cardiovascular risk, but despite considerable improvements in our understanding of HDL cholesterol and its metabolism, none of the pharmacologic agents that raise HDL cholesterol that have been tested so far have had a significant effect on cardiovascular morbidity and mortality. Ongoing clinical trials of agents that raise HDL-cholesterol levels and that have other anti-inflammatory and anti-atherosclerotic effects are currently under way[67].

Genomics, Cell-Based Therapies and Molecular Targeting

Genetic investigations have led to discoveries of the heritable components of cardiovascular risk factors and coronary artery disease[68,69]. Multiple chromosomal loci associated with CAD harbour protein-coding genes known to be important in variations in lipid levels. In addition, associations of single-nucleotide polymorphisms with chemokines suggest that an inflammatory pathway may regulate the process of coronary atherosclerosis[69]. Knowledge of molecular pathways is essential to the development of therapeutics, defined conceptually as "molecular targeting."

Pharmacogenomics applies our understanding of genetic variability in patients' responsiveness to a drug in order to inform clinical decisions about dosing and selection. Genetic variation in CYP2C9 and VKORC1, the two genes that encode the liver proteins required for warfarin metabolism, explains up to 40% of the differences observed among patients in their responses to the same dose of warfarin. In patients with genetic variations in the cytochrome P-450 enzyme, CYP2C19, the antiplatelet drug clopidogrel is less efficacious and the risk of coronary artery disease is increased[70].

Cell-based therapies ranging from autologous noncardiac cells (e.g., bone marrow, skeletal muscle, fat, and endothelial progenitors) to allogeneic mesenchymal cells have been

studied in preclinical animal models and in early trials in humans, with mixed, yet promising, results[71-73].A subset of progenitors is mobilized in vivo by paracrine signals in cases of cardiac injury, suggesting that the delivery of such signals to the heart or vasculature may stimulate regenerative tissue[74].

Global Cardiovascular Disease

CVDs are no longer confined by geographical area or by age, sex or socioeconomic boundaries. Heart disease has already reached epidemic proportions in poorer countries. Of the 45.0 million adult deaths reported worldwide in 2002, three-quarters (32 million) were due to non communicable diseases (WHO 2003)[76]. Except in Africa, non communicable diseases outnumbered communicable diseases in all WHO regions worldwide. In Southeast Asia alone, 7 423 000 deaths were due to non communicable diseases as compared with 5 730 000 deaths related to communicable diseases in the year 2002. Globally, ischemic heart disease (IHD) was the leading killer in the age group $\geq$ 60 years, and, with 1 332 000 deaths in adults aged 15–59 years, IHD was ranked behind HIV/AIDS only.

It is well known that the demographic transition in Western countries was accompanied by a decrease in deaths due to infectious diseases and increased mortality due to noncommunicable diseases. India is in the midst of such demographic transition. The average life expectancy at birth in India is 63.7 years, being 63.1 for males and 64.4 for females [77]. However, this demographic transition has also led to an increase in the number of older people (aged $\geq$60 years), from 19.61 million in 1950 to 75.93 million in 2000 [78]. The increase in life expectancy has brought a large section of the population to an age where CVD starts manifesting itself.

Cardiovascular disease in India

In India, CAD rates have increased during the last 30 years, whereas declining trends have been noticed in developed Western countries[79]. Reports on CAD in Indians from different parts of the world have shown that Asian Indians are at 3–4 times higher risk of CAD than white Americans, 6 times higher than Chinese, and 20 times higher than Japanese[80-83]. The exact prevalence of CAD in India is difficult to estimate owing to the lack of a large prospective study. Absence of a centralized death registry for CVDs and irregularities in completion of death certificates also hamper estimation of the actual burden of CVD[84]. However, various independent epidemiological studies [85,86]conducted in North India suggest that the prevalence of CAD has increased from 1% in 1960 to 10.5% in 1998 in the urban population. A higher prevalence of CAD, ranging from 11.0% to 14.2%, has been reported from South India[87-89]. In rural India, a twofold increase has been reported in the northern states[86,90-94]. A higher prevalence of 7.4% was observed in some parts of rural South India as long ago as in 1993[95]. Taking into account the size of the Indian population, these prevalence rates, translated into figures, indicate that a large number of deaths can be attributed to CAD.

Of particular concern to India is not only the high burden of CVDs, but also the effects of CVD on the productive workforce aged 35–65 years. The incidence of CAD in the young has been reported to be 12%–16% in Indians[19,20]. Half of the CVD-related deaths (52% of CVDs) in India occur below the age of 50 years, and about 25% of acute myocardial infarction (MI) in India occurs under the age of 40 years)[79,96], a finding similar to that of the INTERHEART study[22]. Among the three Asian populations, Chinese, Malay, and Indian, the highest age-standardized incidence rates in both sexes are in Indians. The first MI attack occurs in 4.4% of Asian women and 9.7% of men at age less than 40 years, which is 2- to 3.5

fold higher than in the West European population and is third highest of all the regions studied worldwide[22]. These studies carried out in India and other places suggest that Asians in general and Indians in particular are at increased risk of MI at a younger age (<40 years), irrespective of whether they have migrated to other countries or are resident Asians.

CREATE Registry

This a large prospective registry study conducted in 89 centres from 10 regions and 50 cities in India[97]. 20,937 patients were enrolled. Of the 20,468 patients who were given a definite diagnosis, 12 405 (60·6%) had STEMI. The mean age of these patients was 57·5 years. Patients with STEMI were younger (56·3 years) than were those with non-STEMI or unstable angina (59·3 years). Most patients were from lower middle (52·5%) and poor (19·6%) social classes. The median time from symptoms to hospital was 360 min, with 50 (25–68) minutes from hospital to thrombolysis. 6226 (30·4%) patients had diabetes, 7720 (37·7%) had hypertension and 8242 (40·2%) were smokers.

Treatments for STEMI differed from those for non-STEMI or unstable angina. More patients with STEMI than with non-STEMI were given anti-platelet drugs (98·2% *vs.* 97·4%), angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARB) (60·5% *vs.* 51·2%), and percutaneous coronary interventions (8·0% *vs.* 6·7%). Thrombolytics (96·3% streptokinase) were used for 58·5% of patients with STEMI. Conversely, fewer patients with STEMI than those with non-STEMI or unstable angina were given β blockers (57·5% *vs* 61·9%), lipid-lowering drugs (50·8% *vs* 53·9%) and coronary bypass graft surgery (1·9% *vs* 4·4%).

The 30-day outcomes for patients with STEMI were death (8·6%), reinfarction (2·3%), and stroke (0·7%). Outcomes for those with non-STEMI or unstable angina were better: death (3·7%), reinfarction (1·2%) and stroke (0·3%). Use of key treatments also

differed by socioeconomic status. More rich patients than poor patients were given thrombolytics (60·6% *vs* 52·3%), β blockers (58·8% *vs* 49·6%), lipid-lowering drugs (61·2% *vs* 36·0%), ACE inhibitors or ARB (63·2% *vs* 54·1%), percutaneous coronary intervention (15·3% *vs* 2·0%) and coronary artery bypass graft surgery (7·5% *vs* 0·7%). Mortality was higher for poor patients than for rich patients (8·2% *vs* 5·5%)[97]. Adjustment for treatments (but not risk factors and baseline characteristics) eliminated this difference in mortality.

Risk factors of premature CAD in Indians

Most of the knowledge on CAD risk factors in different age groups is from studies carried out in the migrant Indian population[98-101], and these have their limitations. It has been suggested that the results of these studies cannot be generalized, as there is either over representation of certain communities or under-representation or absence of other communities.

Heart diseases are occurring in Indians 5 to 10 years earlier than in other populations around the world[80,102]. According to the INTERHEART study[22], the median age for first presentation of acute MI in the South Asian (Bangladesh, India, Nepal, Pakistan, Sri Lanka) population is 53 years, whereas that in Western Europe, China, and Hong Kong is 63 years, with more men than women affected. Data from the Singapore Myocardial Infarction Registry from 1988 to 1997 for acute MI cases aged between 20 and 64 years also showed that men were four times more prone to these events than women. The median age for presentation of first MI was higher in Asian women than in Asian men (58 and 54 years, respectively), a finding similar to that of the INTERHEART study[22]. It must be emphasized that although the median age of presentation is higher in women, they are known worldwide to have poor prognosis compared with men[103-107]. Younger Asian women have worse survival

at 28 days after acute MI. The reasons for higher mortality in younger women are poorly understood and may be related to the presence of different risk factors in women, co morbidities, severity of infarction, and response to treatment. In view of the above discussion, it is imperative to ascertain the causes of the rising prevalence and emergence of CAD earlier in the life of Indians.

The **INTERHEART study**[22], involving 52 countries, established an association between conventional modifiable risk factors for MI in all regions of the world, including South Asia, and in both sexes and at all ages. In South Asians, apolipoprotein (Apo)B/ApoA1 (odds ratio [OR] 3.81) and smoking (OR 2.43) were the important risk factors, as in the rest of the world. However, hypertension (OR 2.89), abdominal obesity (OR 2.43), and diabetes (OR 2.48) had more severe effects in South Asia, whereas psychosocial factors had an odds ratio of 2.15, compared with 2.67 worldwide. The INTERHEART study[22] also showed that hypertension and diabetes were more important risk factors in younger Indian women than men. Earlier studies, mostly in Western populations, have also found an association of the above-mentioned risk factors with the development of CAD. For example, in the Prospective Cardiovascular Munster Heart Study (PROCAM), a large prospective study in men aged 35–65 years, eight variables that made an independent contribution to risk of CAD were age, systolic blood pressure, LDL-C, HDL-C, triglycerides, diabetes mellitus, smoking and family history of MI[108]. The Jaipur Heart Watch-2 study is a prospective study from North India that found a high prevalence of classical risk factors for CAD, namely smoking, low physical activity, hypertension, hypercholesterolemia, diabetes, and obesity in urban men and women of Jaipur[109]. Factors peculiar to the South Asian population, such as truncal obesity, low HDL-C, and high triglycerides, were also prevalent. A significant increase in people with obesity, diabetes, and dyslipidemia was observed as compared with those reported in the Jaipur Heart Watch-1 study carried out in the early 1990s in the same population. An

increasing prevalence of impaired glucose tolerance and diabetes in urban residents of Chennai has also been reported[110]. Fewer studies on epidemiological data from angiographically proven cases of premature CAD (≤40 years) in native Indians are available[111-117]. Hyperlipidemia was found to be prevalent in young Indians with CAD in these studies. However, differences appear to exist between the lipid levels present in North and South Indian CAD patients and individuals without CAD. It appears that North Indians manifest the disease at lower levels of total cholesterol. Also, a greater role can be attributed to total cholesterol and LDL-C in atherogenesis in the younger Indian population (≤40 years) with angiographically proven CAD. The lower HDL-C and higher triglyceride levels in both younger and older cases appear to be a hallmark of the Indian population[89,117]. In the INTERHEART study[22] also, the highest population attributable risk (PAR) was abnormal lipids (ApoB/ApoA1 ratio) in both sexes. These studies indicate that abnormalities in lipid metabolism play an important role in development of CAD in young Indians. Also, compared with women[113], young Indian male patients have a slightly lower prevalence of hypertension and diabetes[,112,117].

Smoking and low physical activity in Indians have been found to be prevalent among 20–39 years old urban adults[109]. The INTERHEART study[22] also observed that smoking was a greater risk factor in younger men than in women. The risk of CAD increased incrementally with smoking. The odds ratio was 9.16 in individuals who smoked more than 40 cigarettes per day, compared with 1.38 in those smoking 1–5 cigarettes per day, indicating that there is no safe limit for smoking. Other epidemiological studies from India also suggest a greater association of smoking with CAD in younger individuals[114,117]. Furthermore, the prevalence of smoking in South Indian males (44.6%) and passive smoking in South Indian females (45.3%) has been reported to be significantly higher than in North Indians[87]. Interestingly, smoking has not been found to be a significant risk factor in acute MI patients from rural

parts of India. The patients from rural India, however, have elevated blood glucose and abnormal waist/hip ratio[118].

Another important independent risk factor for CAD in younger cases emerging out of Indian studies is family history of CAD[111-114]. The INTERHEART study[22] showed a population attributable risk (PAR) of 14.8% in younger versus 10.45% in older patients. Though addition of family history of CAD to other risk factors causes only a modest increase in PAR by 1%, it must be emphasized that modifiable physiological variables such as blood pressure, ApoB/ApoA1 ratio, serum cholesterol, and abdominal obesity are also partially under genetic control. As family history of CAD emerged as the second most important risk factor in young Indian patients[117], analysis of potential genetic factors such as variance of genes involved in vascular homeostasis, hemostatic factors, lipid metabolism, and other metabolic factors is warranted.

Evidence from studies carried out in South Asians suggests that conventional risk factors account for 90% of the PAR in men and 94% in women (hypertension, smoking, diabetes, ApoB/ApoA1 ratio and abdominal obesity accounted for 80.2% of the PAR). As these risk factors were also established by Indian studies to be involved in premature CAD, there are now enough data for establishment of health policies to counteract the threat posed by the epidemic of CVDs in India. Modification of environmental triggers for these risk factors is likely to reduce the speed of this advancing epidemic. The benefits of genetic research in this area can in future be reaped in economic terms.

Diabetes Mellitus and CAD

Diabetes mellitus is a group of diseases characterized by insufficient production of insulin or by the failure to respond appropriately to insulin, resulting in hyperglycemia. The

diagnostic criteria are summarized in Table 1.[122] Importantly, new to the diagnostic criteria in 2010, a glycosylated hemoglobin (A1c) level ≥6.5% has been added. Diabetes is typically classified as type 2 diabetes, characterized by relative insulin deficiency with a backdrop of insulin resistance and representing >90% of all diabetes cases, or type 1 diabetes, characterized by absolute insulin deficiency.

Diabetes is among the most common chronic diseases in the world, affecting an estimated 180 million people in 2008.[123] Confounding this high global burden is the increasing incidence and prevalence of type 2 diabetes, driven by increasing population age, obesity and physical inactivity as well as by the increasing longevity of patients with diabetes. Estimates project that more than 360 million persons will be affected by diabetes by 2030.

Table 1. American Diabetes Association Diagnostic Criteria for Diabetes Mellitus[122]

Fasting plasma glucose ≥ 7.0 mmol/liter (126 mg/dL)
or
2-hour plasma glucose ≥ 11.1 mmol/liter (200 mg/dL) during standardized 75-g oral glucose tolerance test
or
Symptoms of hyperglycemia plus nonfasting plasma glucose ≥ 11.1 mmol/liter
or
A1c ≥ 6.5% (200 mg/dL)

Whereas much attention historically has focused on the prevention and treatment of microvascular disease complications of diabetes, cardiovascular disease (CVD) remains the principal morbidity and driver of mortality in the setting of diabetes, most commonly in the

form of coronary heart disease (CHD), cerebrovascular disease, peripheral vascular disease, and heart failure. For these reasons, continual efforts toward mitigating the risk of CVD in diabetes remain a global public health imperative.

Compared with nondiabetic individuals, patients with diabetes have a 2-4 times increased risk for development and dying of CHD[124]. Whereas older studies have suggested a diabetes associated CVD risk similar to that observed among nondiabetic patients with MI, that is, a "coronary disease equivalent", more recent observations from clinical trials suggest a substantially lower CHD risk, most likely reflecting the effectiveness of contemporary therapeutic interventions.[125-7]

Diabetes is associated with an increased risk for MI. Across the spectrum of ACS events, in which diabetes may affect more than one third of patients,[128] patients with diabetes have worse CVD outcomes.[129] Despite overall improvements in outcomes during the past several decades for ACS, the gradient of risk associated with diabetes persists.[129] Furthermore, the increased risk observed with diabetes in the setting of ACS events extends to glucose values in the range well below the diabetes threshold, whether it is analyzed by glucose values at the time of presentation or those observed throughout hospitalization.[130] Compared with nondiabetics, diabetic patients have a greater atherosclerotic burden, both in the major arteries and in microvasculature. Diabetic patients have substantially increased rates of atherosclerotic complications in the settings of primary prevention and after coronary intervention procedures.

Prediabetes and CAD

Almost 35 million Americans have some degree of abnormal glucose tolerance, a condition along with obesity that markedly increases the risk for type 2 diabetes and premature atherothrombosis. Insulin resistance alone confers an elevated risk of heart failure and probably explains the association of obesity with this diagnosis.[131] Moreover, the risk of cardiovascular disease starts to increase long before the onset of clinical diabetes. In the Nurses Health Study, women who eventually developed type 2 diabetes had a threefold elevated relative risk of myocardial infarction before the diagnosis of diabetes, a cardiovascular event rate almost as high as that in patients with overt diabetes at study entry.[132] These effects are magnified in ethnic minority populations and in patients with other concomitant risk factors.

Although hyperglycemia associates with microvascular disease, insulin resistance itself promotes atherosclerosis even before it produces frank diabetes, and available data corroborate the role of insulin resistance as an independent risk factor for atherothrombosis. This finding has prompted recommendations for increased surveillance for the metabolic syndrome, a cluster of glucose intolerance and hyperinsulinemia accompanied by hypertriglyceridemia, low HDL levels, hypofibrinolysis, hypertension, microalbuminuria, predominance of small, dense LDL particles, and central obesity.

Although several formal definitions of the metabolic syndrome have been proposed, the definition adopted by the National Cholesterol Education Program Adult Treatment Panel requires at least three of the following five criteria:

1. Waist circumference larger than 102 cm in men and 88 cm in women

2 .Serum triglyceride levels of at least 150 mg/Dl

3. HDL cholesterol less than 40 mg/dL in men and less than 50 mg/dL in women

4. Blood pressure of at least 130/85 mm Hg

5.Serum glucose concentration of at least 110 mg/dL.

Table 2. Mechanisms Implicated in Diabetic Vascular Disease

Endothelium	↑ NF-κβ activation ↓ Nitric oxide production ↓ Prostacyclin bioavailability ↑ Endothelin 1 activity ↑ Angiotensin II activity ↑ Cyclooxygenase 2 activity ↑ Thromboxane A_2 activity ↑ Reactive oxygen species ↑ Lipid peroxidation products ↓ Endothelium-dependent relaxation ↑ RAGE expression
Vascular smooth muscle cells and vascular matrix	↑ Proliferation and migration into intima ↑ Increased matrix degradation Altered matrix components
Inflammation	↑ IL-1β, IL-6, CD36, MCP-1 ↑ ICAMs, VCAMs, and selectins ↑ Activity of protein kinase C ↑ AGEs and AGE/RAGE

AGEs - advanced glycation end products; ICAMs - intracellular adhesion molecules; IL - interleukin; MCP - monocyte chemoattractant protein; NF - nuclear factor; RAGE - receptor for advanced glycation end products; VCAMs - vascular cell adhesion molecules.

Hyperglycemia is likely to directly influence atherosclerosis development, progression, and instability. The principal vascular perturbations linked to hyperglycemia include endothelial dysfunction, vascular effects of advanced glycation end products, adverse effects of circulating free fatty acids, and increased systemic inflammation. In addition, the pernicious effects of hypoglycemia complicating diabetes therapy, the sympathovagal imbalance due to diabetic autonomic neuropathy, and the vascular effects of constitutive exposure to excess insulin may further contribute to atherosclerotic risk.

Endothelial dysfunction, a hallmark of diabetic vascular disease, is associated with increased hypertension and adverse CVD outcomes. The myriad mechanisms contributing to endothelial dysfunction include abnormal nitric oxide biology, increased endothelin and angiotensin II, and reduced prostacyclin activity, all of which contribute to abnormal control of blood flow. In the setting of ACS events, no-reflow after percutaneous intervention reflecting acute endothelial dysfunction occurs more commonly in the presence of diabetes or hyperglycemia and may contribute to increased myocardial jeopardy, resulting in larger infarcts, increased arrhythmia, and worse systolic function.

Diabetic dyslipidemia is characterized by high triglyceride levels, low HDL and increased atherogenic small dense LDL particles, each of which is likely to contribute to the accelerated development and progression of atherosclerosis. Perturbations in the fibrinolytic system and platelet biology further compound the direct vascular effects of diabetes, yielding a constitutive prothrombotic milieu. These abnormalities include increased circulating tissue factor, factor VII, von Willebrand factor, and plasminogen activator inhibitor 1, with decreased levels of antithrombin III and protein C. In addition, disturbances of platelet activation, aggregation, morphology and life span further contribute to increased thrombotic potential, as well as to the acceleration of atherosclerosis.

Table 3. Perturbations of Platelet Function Associated with Diabetes

Reduced membrane fluidity
Altered Ca^{2+} and Mg^{2+} homeostasis
Increased arachidonic acid metabolism
Increased thromboxane A_2 synthesis
Decreased nitric oxide and prostacyclin production
Decreased antioxidant levels
Increased expression of activation-dependent adhesion molecules (e.g., glycoprotein IIb/IIIa, P-selectin)
Increased platelet micro particle formation
Increased platelet turnover

Increased systemic inflammation portends an increased risk for diabetes and diabetic atherosclerotic disease, and diabetes is associated with increased oxidative stress and the accumulation of advanced glycation end products. Diabetes is associated with lipid-rich atherosclerotic plaque and increased inflammatory cell infiltration, increased expression of tissue factor, and increased expression of the receptor for advanced glycation end products, yielding plaques with characteristics of higher risk in both coronary and carotid arteries.

Prediabetes and CAD -Evidence

A meta-analysis[133] showed that the estimated relative risk (RR) for cardiovascular disease associated with impaired glucose tolerance (IGT) ranges from 0.97 to1.30 and that associated with impaired fasting glucose (IFG) ranges from approximately1.12 to 1.37. Furthermore, the risk associated with IFG 110 mg/dl was larger than that for IFG 100 mg/dl. At present, the available data are insufficient to confirm the presence of sex difference in the risk between pre-diabetes and cardiovascular disease.

Some reviews have suggested that IGT increased the risk for macrovascular disease by approximately 2-fold. Subsequent studies that were based on the 1980 or 1985 WHO criteria in which IGT was defined as a fasting plasma concentration of glucose of 140 mg/dl and a 2-h concentration of glucose of 140 to 200 mg/dl also reported an approximate doubling of risk for cardiovascular disease among participants with IGT. Although more data were available for IFG 110 mg/dl than for IFG 100 mg/dl, the number of such studies was still limited.

Regarding IGT, there are currently insufficient data to arrive at a conclusion concerning potential sex differences. Of note is the finding from the DECODE study in 2001 that the RRs for cardiovascular disease among participants with 2-h glucose abnormalities corresponding to IGT were very similar for men and women[134]. It is not clearly known whether the risk for developing cardiovascular disease is present for people with pre-diabetes even if they never develop diabetes. At least 2 attempts have been made to address this issue and have failed to produce definitive insights into this issue[135].

Current recommendations to screen for Prediabetes are inconsistent. The U.S. Preventive Services Task Force does not support screening for pre-diabetes, whereas the ADA supports screening among people at increased risk on the basis of age and body mass index. Nevertheless, an economic analysis that incorporates the prevention of CVD might

provide additional useful information concerning the need to screen for pre-diabetes in the general population or in specific population groups at high risk.

In summary, the exact magnitude of the risk for CVD associated with IFG or IGT remains opaque at present. Depending on the set of studies examined, analyses could be interpreted as implying no increase in risk or at most a very modest increase in risk. Given the sizeable and growing percentage of adults who have pre-diabetes in some countries like the United States, South Asia, a small increase in risk might still translate into substantial numbers of adults developing or dying from cardiovascular disease.

Prediabetes in India

The metabolic syndrome (MS) is common among South Asians. The term South Asian refers to all individuals who have ancestral origin in the Indian subcontinent (India, Pakistan, Bangladesh, Nepal, and Srilanka). Because South Asians develop metabolic abnormalities at a lower body mass index (BMI) and waist circumference(WC) than other groups, conventional criteria underestimate the prevalence of MS by 25% to 50%. The South Asian Modified National Cholesterol Education Program criteria that use abdominal obesity as an optional component and the South Asian specific waist circumference recommended by the International Diabetes Federation appear to be more appropriate in this population. Furthermore, Asian Indians have at least double the risk of CAD than that of whites, even when adjusted for the presence of diabetes and MS. This increased risk appears to be due to South Asian dyslipidemia, which is characterized by high serum levels of apolipoprotein B, lipoprotein (a), triglycerides and low levels of apolipoprotein A1 and HDL cholesterol. In addition, the LDL particles are small, dense, and dysfunctional. MS needs to be recognized as a looming danger to South Asians and treated with aggressive lifestyle modifications beginning in childhood and at a lower threshold than in other populations.

Asian Indian or South Asian Phenotype

Many Asian Indians fit into the model of metabolically obese, normal weight individuals.[136] This group comprises only 6% of all whites but a substantial segment of Asian Indians.[137] Many Asian Indians develop diabetes and MS with a BMI <25 kg/m2, which is generally considered normal among whites. South Asians, in general, and Asian Indians, in particular, have certain unique clinical and biochemical characteristics that are collectively referred to as the "South Asian" or "Asian Indian" phenotype. Compared with whites at comparable BMI and age, Asian Indians have profoundly higher dyslipidemia, and hypoadiponectinemia, greater WC, thinner hips, short legs and increased cardiovascular risk[138]. For any given WC, they also have increased visceral fat and insulin resistance that are evident even among children aged 8 to 11 years[139]. For example, South Asian children with a WC of 80 cm have higher insulin levels than white children with a WC of 90 cm. In addition, South Asians also have significant procoagulant tendencies as shown by high plasminogen activator inhibitor-1and fibrinogen concentrations.[140] These metabolic abnormalities also contribute to the increased predilection for diabetes and CAD.

Differing Criteria for Obesity and Abdominal Obesity among Asians

Because of the above mentioned observations, The World Health Organization(WHO) has issued a lower cut-off point for overweight (BMI >23) and obesity (BMI >25) for all Asians.[141] By this criterion, 95% of South Asian diabetic patients were identified as overweight and 80% were obese in the United Kingdom Asian Diabetes Study(UKADS)[142]. A more recent study found WC of 87 cm for men and 82 cm for women as appropriate cut-off points to identify cardio metabolic risk factors including prediabetes in urban Asian Indians[143].35 The WHO and IDF have also issued lower cut-off points for WC for the diagnosis of abdominal obesity for South Asian men (90 cm) and women (80 cm)[144].

South Asian Modified NCEP Criteria for MS

Although NCEP does not provide ethnic-specific cut-off points for waist circumference (WC), the 2005 AHA/NHLBI Scientific Statement on MS endorses the lower WC for all Asian Americans (<90 cm for men and <80cm for women).[145] The South Asian Modified(SAM) NCEP Criteria follows the NCEP criteria for MS except for the inclusion of South Asian specific WC cut-off points for abdominal obesity as recommended by the IDF.[145-6] Thus, unlike the IDF criteria, abdominal obesity is considered optional, not essential. Among South Asians, the prevalence of MS is higher by 30% to 50% when SAM-NCEP criteria are applied compared with NCEP criteria and 20% higher compared with IDF criteria. Clinical diabetes and CAD are preceded by a constellation of risk factors that are also the components of MS, the prevalence of which among Asian Indians is approximately 25% with either NCEP or IDF criteria. The prevalence increases to 35% to 40% when the SAM−NCEP criteria are used. The prevalence of MS among South Asians is higher than in other Asians and Europeans. The syndrome confers a 2-fold risk of CAD and a 5-fold risk of diabetes.

Primary treatment of MS is lifestyle therapy and includes weight loss, increased physical activity, and an antiatherogenic diet. Adopting a healthy lifestyle beginning in childhood and adolescence is warranted in view of the malignant nature of CAD among Asian Indians. Because the adverse effects of these factors are greater in Indians, the benefits of modifying the factors are correspondingly greater and may prevent the onset of diabetes.

AIMS OF THE STUDY

AIMS OF THE STUDY

The primary aims of the study are to analyze

1. The prevalence of Conventional risk factors among patients who present with Myocardial infarction at young age.
2. The demographic profile of patients.
3. The prevalence of Hyperhomocysteinemia and high hemoglobin.

The secondary aims of the study are

1. To analyze the mode of presentation.
2. To analyze the profile of coronary lesions in angiogram.
3. To assess the prevalence of LV dysfunction.

MATERIALS AND METHODS

MATERIALS AND METHODS

This is a prospective observational study of young patients admitted with a diagnosis of myocardial infarction to our Intensive Coronary Care Unit and evaluated.

Young patients with age 40 years or less were included in the study. Most similar studies of premature CAD have used an age cut-off of 40 to 45 years to define "young" patients with CAD and AMI.[1-5]. The study was conducted over a period of one year from 1st October 2010 to 30th September 2011. All consecutive patients admitted with Acute coronary syndromes (ACS), both STEMI and NSTEMI as per standard definition during this period were included in the study after consent. The study was conducted at PSG hospitals, the teaching affiliate of PSG institute of Medical Sciences and research (PSG IMS & R) which is a teaching and tertiary care referral hospital located in the city of Coimbatore. Patients are from the city as well as from nearby towns, villages and a few from the adjoining state of Kerala.

The patients who did not get the complete evaluation including coronary angiogram for various reasons were excluded from the study.

The purpose of the study was explained to the patients and relatives and consent was obtained. The study was approved by the Human Ethics Committee of the institution before commencement of the study.

The Inclusion criteria were

1. Age less than or equal to 40 years.
2. Both male and female patients.
3. Typical chest pain suggestive of AMI.
4. ST elevation in ECG as per definition at the time of presentation with chest pain (STEMI)

5. Positive Troponin T (NSTEMI)

Exclusion criteria:

1. Age > 40 years.
2. Non cardiac chest pain.
3. Patients who did not get admitted.
4. Patients who did not undergo coronary angiogram.

All Patients' demographic profile, income and marital status were noted at the time of admission.

Patients were enquired for standard history of hypertension, diabetes mellitus, dyslipidemia, smoking, alcohol consumption and family history of Coronary Artery Disease.

Blood investigations at the time of admission included complete blood count, fasting lipid profile, Hb A1c.

Patients were labeled as hypertensive if they have been documented to have high Blood Pressure during previous medical consultations, if they are on antihypertensive medications, or if their BP is $\geq$ 140/90 consistently during current admission.

Patients were labeled as diabetic if they have documented fasting blood sugar of 127 mg/dl or Hb A1c of > 6.5 gm% or if the same is noted during current admission. Fasting serum Homocysteine level and hemoglobin levels were checked in all patients.

All patients underwent transthoracic 2D and Doppler echocardiogram with colour Doppler study. LV systolic and diastolic function were routinely assessed. Mechanical complications like Mitral regurgitation, ventricular septal rupture, free wall rupture and pericardial effusion were also looked for.

Patients underwent treatment modalities of Thrombolysis, Primary Percutaneous Coronary Intervention (PCI) or conservative management as feasible and required, depending on the clinical circumstances and consent at the time of presentation. All patients were treated with dual antiplatelets, atorvastatin, low molecular weight heparin and other indicated drugs as per standard protocols.

All patients underwent coronary angiogram either at the time of admission for primary PCI or electively after initial medical stabilization with Thrombolysis or conservative treatment. Presence of coronary lesion, type of lesion, presence or absence of thrombosis, myocardial bridging, coronary anomalies and ectasia were looked for.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

69 PATIENTS WERE INCLUDED FOR THE STUDY BASED ON THE ENTRY CRITERION.

Age

Among the 69 patients studied, 6 patients were under the age of 30 years (8.7%). Remaining 63 years patients were between 31-40 years of age (91.3%).

Sex

60 Patient were Males (86.95 %), 9 patients were Females (13.05 %), which is statistically significant. 63 Patients (91.3%) were married. 6 patients (8.7%) were unmarried.

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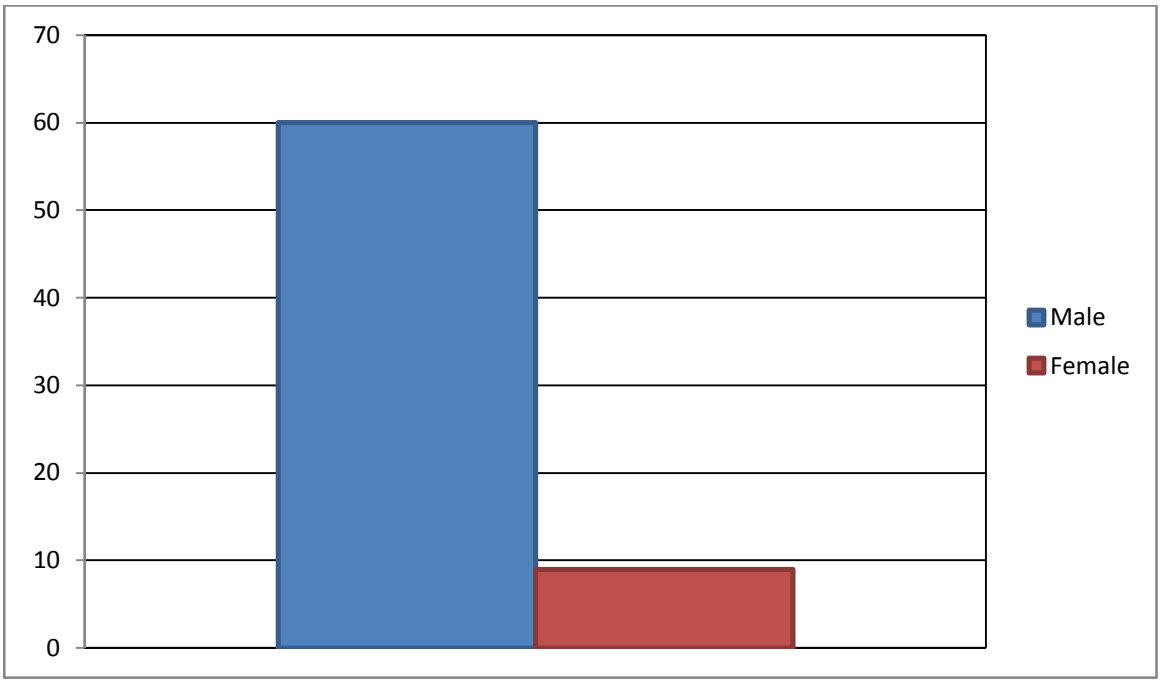
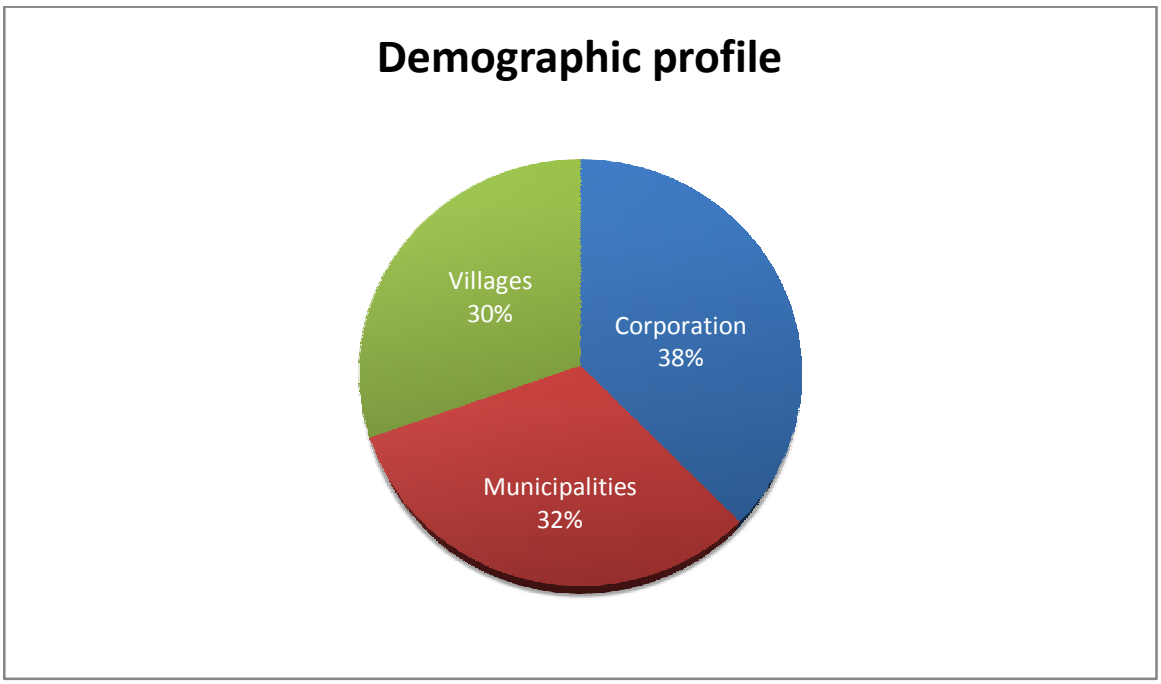


Bar diagram showing proportion of male and female patients

Demographic profile

When residence of the patients was analyzed, 26 patients (37.7%) came from corporation (Cities, Big towns), 22 patients (31.9%) came from municipalities and 21 patients (30.4%) came from villages.



According to income, patients were divided into four groups. 32 patients (46.42%) were earning less than Rs. 72000 per annum, 24 patients [34.8 %] were earning between Rs.72000-2,00,000, 6 patients (8.7%) were earning Rs.2,00,000- 4,00,000 and 7 Patients (10.1%) were earning > Rs. 4,00,000 per annum.

Conventional risk factors

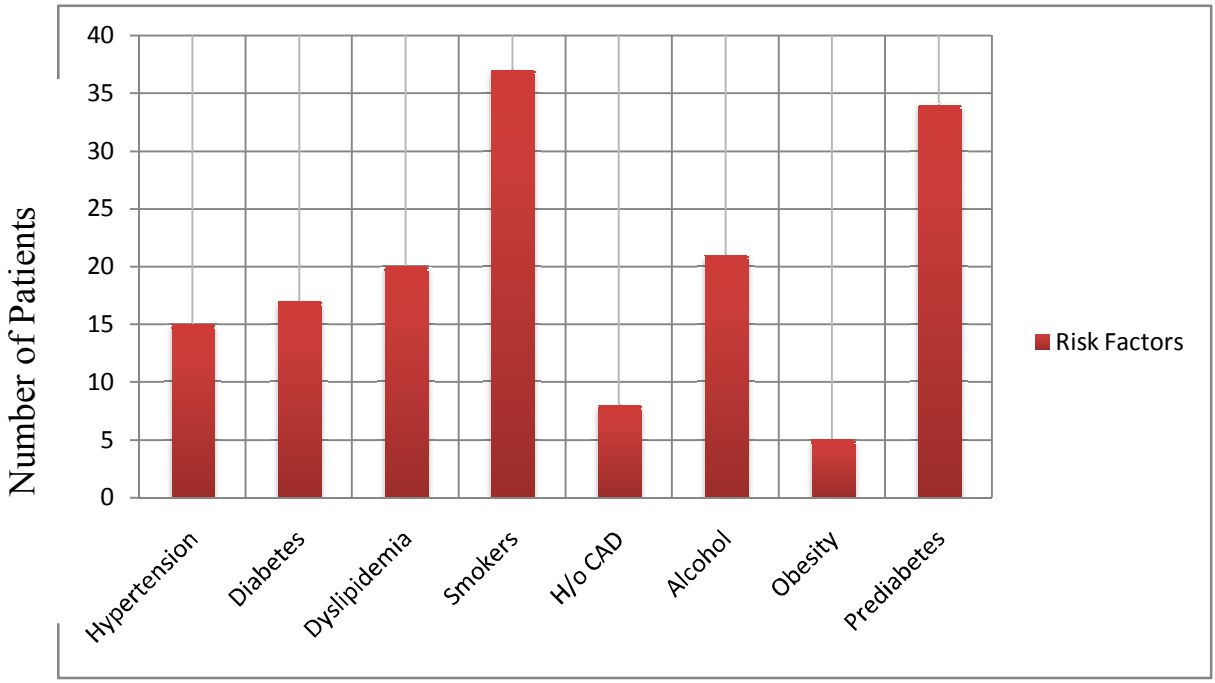
1. Hypertension was present in 15 patients (21.7%).
2. 17 Patient (24.6%) were diabetics, among whom 2 of them were Insulin requiring.
3. 20 Patient (29 %) had history of dyslipidemia.
4. 37 patients (53.6%) were current smokers or have been smoking in the recent past.
5. 8 patients (11.6%) had family H/o Coronary artery disease.
6. 21 patients (30.4%) were consuming alcohol on a regular basis.
7. 5 patients (7.2%) had obesity based on BMI.
8. Patients were labeled as having pre diabetes if their HbA1C is between 5.5 and 6.4 in the absence of documented Diabetes mellitus in the past. 34 patients (49.27%) had prediabetes. 20 patients (28.98%) had HbA1C < 5.5 gm%, 15 patients (21.75%) had HbA1C > 6.5 gm% .

Lipid profile

All patients had fasting lipid profile done during admission within 48 hours.

1. 54 Patients (78.26%) had LDL cholesterol > 100mg%.
2. 53 Patients (76.82 %0 had HDL< 40mg %.
3. 35 Patients (50.72%) had TGL level > 150%.

RISK FACTORS



Other factors

1. **Vasculitis** : 2 patients (2.9%) had H/o symptoms of suggestive of vasculitis, both of whom are female patients.
2. **High Hemoglobin** : Hemoglobin was checked in all patients. 13 patients (18.84%) had Hb <13, among whom 2 patients (2.9%) had Hb < 10 gm%. 5 patients (7.25%) had Hb > 17gm%.
3. **Hyperhomocysteinemia**: 13 patients(18.8 %) had hyperhomocysteinemia whose levels were from 15 to more than 50 micromol/lit.

Mode of presentation

According to ECG analysis, 50 patients (72.5%) had STEMI, 19 patients (27.5 %) had NSTEMI.

Left ventricular function

All patients underwent transthoracic echocardiogram. Mild LV dysfunction is defined as EF 40- 49 %, moderate LV dysfunction as EF 30-39 %, severe LV dysfunction as < 30 %. Majority, 54 patients (78.25%) had normal LV function. 13 patients (18.95%) had mild LV dysfunction, 2 patients (2.9%) had moderate LV dysfunction (EF 31-45%). None of them had severe LV dysfunction.

Complications

1. 3 patients (4.3%) presented with recurrent MI.
2. 2 patients (2.9%) developed arrhythmias both of which were ventricular arrhythmias.
3. None of them developed Pericardial effusion or any significant mechanical complications.
4. None of them had atrial fibrillation.
5. No incidence of cerebrovascular accident was observed.
6. None of the patients died during the index admission.

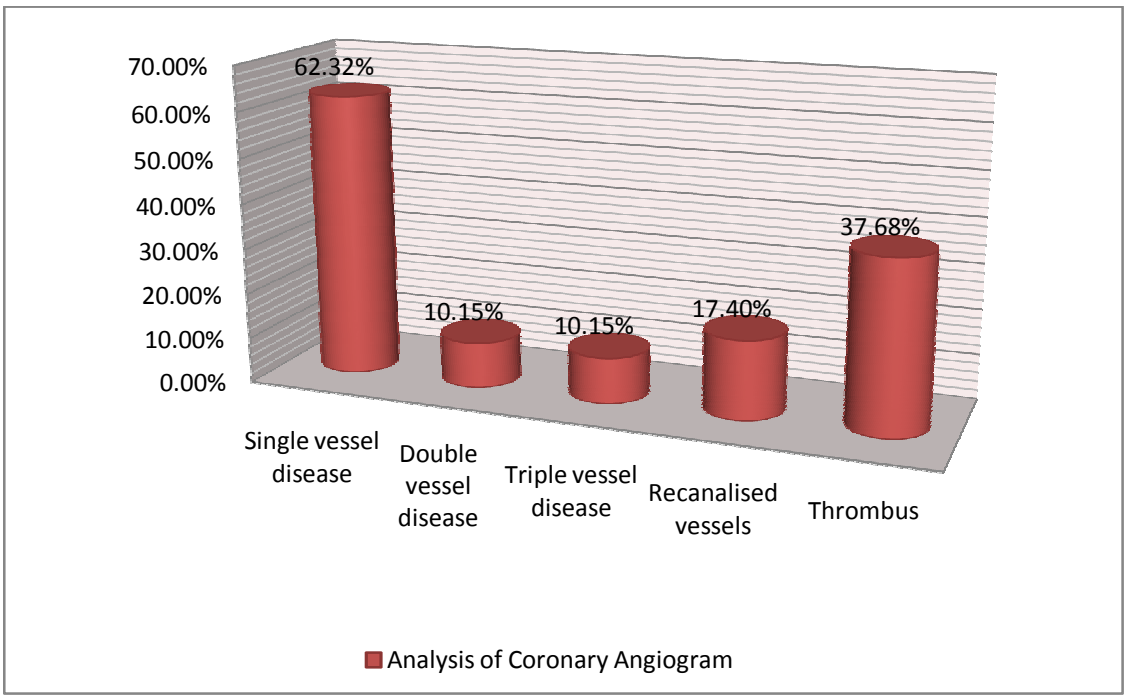
Treatment

Among the 50 patients who had STEMI, 22 patients underwent thrombolysis with streptokinase (44 %) and 13 patients (26 %) underwent primary PCI. 15 patients (30 %) were treated conservatively with conventional treatment.

Coronary angiogram

Analysis of coronary angiogram showed

1. Single vessel disease in 43 patients [62.32 %]
2. Double vessel disease in 7 patients [10.15 %]
3. Triple vessel disease in 7 patients [10.15 %]
4. Recanalised vessels in 12 patients [17.4 %]
5. Thrombus was seen in 26 patients [37.68%].
6. Collateral vessels, bifurcation lesion, coronary ectasia were seen in one patient each.



Among the single vessel disease patients, Left Anterior Descending artery or its major branch involvement was the most common presentation seen in 33 patients (47.8 %) followed by right coronary artery involvement in 6 patients (8.7 %). Left Circumflex artery was the least common infarct related artery, seen in 4 patients (5.8 %).

Residence based analysis:

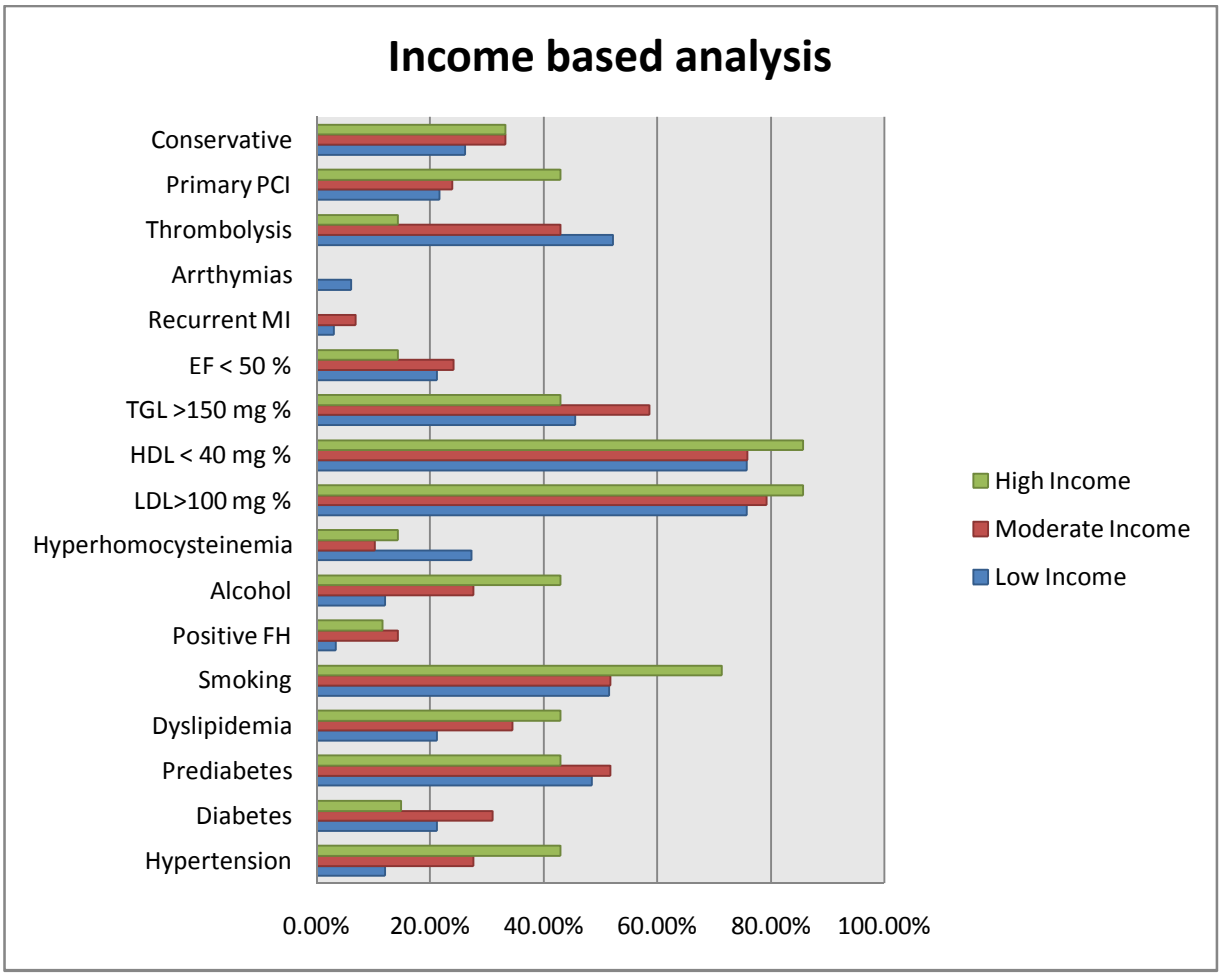
	Urban	Rural
Total	48 [69.56 %]	21 [30.44 %]
Hypertension	9 [18.8 %]	**6 [28.6 %]**
Diabetes	**14 [29.2 %]**	3 [14.3 %]
Prediabetes	20[41.6 %]	12[57.2 %]
H/o Dyslipidemia	14 [29.2 %]	6 [28.6 %]
Smoking	19 [39.6 %]	**18 [85.7 %]**
Positive Family history	5 [10.4 %]	3[14.3 %]
Vasculitis	2	0
Alcohol consumption	16 [33.3 %]	5[23.8 %]
hyperhomocysteinemia	9[18.8 %]	4[19 %]
LDL > 100 mg %	**38 [79.2 %]**	5[23.8 %]
HDL < 40 mg %	37[77 %]	16 [76.2 %]
TGL > 150 mg %	26[54.2 %]	9[42.9 %]
Hb > 17 gm %	4[8.3 %]	1 [4.8 %]
LV Dysfunction [EF < 50 %]	11[22.9 %]	4[19 %]
Recurrent MI	3 [6.2 %]	0
Arrythmias	2 [4.2 %]	0
STEMI	36[75 %]	14[66.6 %]
NSTEMI	12[25 %]	7[33.3 %]
Thrombolysis	15[41.66 %]	7[50 %]
Primary PCI	**12[33.3 %]**	1[7.14 %]

When urban and rural patients were compared, urban patients had higher incidence of diabetes(29.2% vs 14.3%), increased LDL(79.2 % VS 23.8 %). Rural patients had higher incidence of hypertension (28.6 % vs 18.8 %), smoking (85.7 % vs 39.6 %). Urban patients underwent primary PCI more frequently than rural patients (33.3 % vs 7.14 %).

Income based analysis

	LOW INCOME	MODERATE INCOME	HIGH INCOME
Total	33	29	7
Hypertension	4[12.1%]	8[27.6%]	3[42.9 %]
Diabetes	7[21.2 %]	9[31 %]	1[14.9 %]
Prediabetes	**16[48.5 %]**	**15[51.7 %]**	**3[42.9 %]**
Dyslipidemia	7[21.2 %]	10[34.5 %]	3[42.9 %]
smoking	17[51.5 %]	15[51.7 %]	5[71.4 %]
Positive FH	6[3.4 %]	1[14.3 %]	1[11.6 %]
Alcohol	4[12.1 %]	8[27.6 %]	3[42.9 %]
hyperhomocysteinemia	9[27.3 %]	3[10.3 %]	1[14.3 %]
LDL > 100 mg %	**25[75.8 %]**	**23[79.3 %]**	**6[85.7 %]**
HDL < 40 mg %	**25[75.8 %]**	**22[75.9 %]**	**6[85.7 %]**
TGL >150 mg %	**15[45.5 %]**	**17[58.6 %]**	**3[42.9 %]**
EF < 50 %	7 [21.2 %]	7[24.1 %]	1[14.3 %]
RECURRENT MI	1[3 %]	2[6.9 %]	0
Arrhythmias	2[6.1 %]	0	0
STEMI	23[69.7 %]	21[72.4 %]	6[85.7%]
NSTEMI	10[30.3 %]	8[27.6 %]	1[14.3 %]
Thrombolysis	12[52.2 %]	9[42.9 %]	1[16.6 %]
Primary PCI	5[21.7 %]	5[23.8 %]	**3[50 %]**
Conservative	6[26.1 %]	7[33.3 %]	2[33.3 %]

Incidence of Prediabetes is high irrespective of the income of the patients, which is 48.5 %, 51.7 %, 42.9 % in low, moderate, high income patients respectively. Incidence of dyslipidemia is also equally high among patients with various income. High income patients underwent primary PCI more frequently [50 %].


Income based analysis
Conservative
Primary PCI
Thrombolysis
Arrthymias
Recurrent MI
EF < 50 %
TGL >150 mg %
HDL < 40 mg %
LDL>100 mg %
Hyperhomocysteinemia
Alcohol
Positive FH
Smoking
Dyslipidemia
Prediabetes
Diabetes
Hypertension
0.00%
20.00%
40.00%
60.00%
80.00%
100.00%
High Income
Moderate Income
Low Income

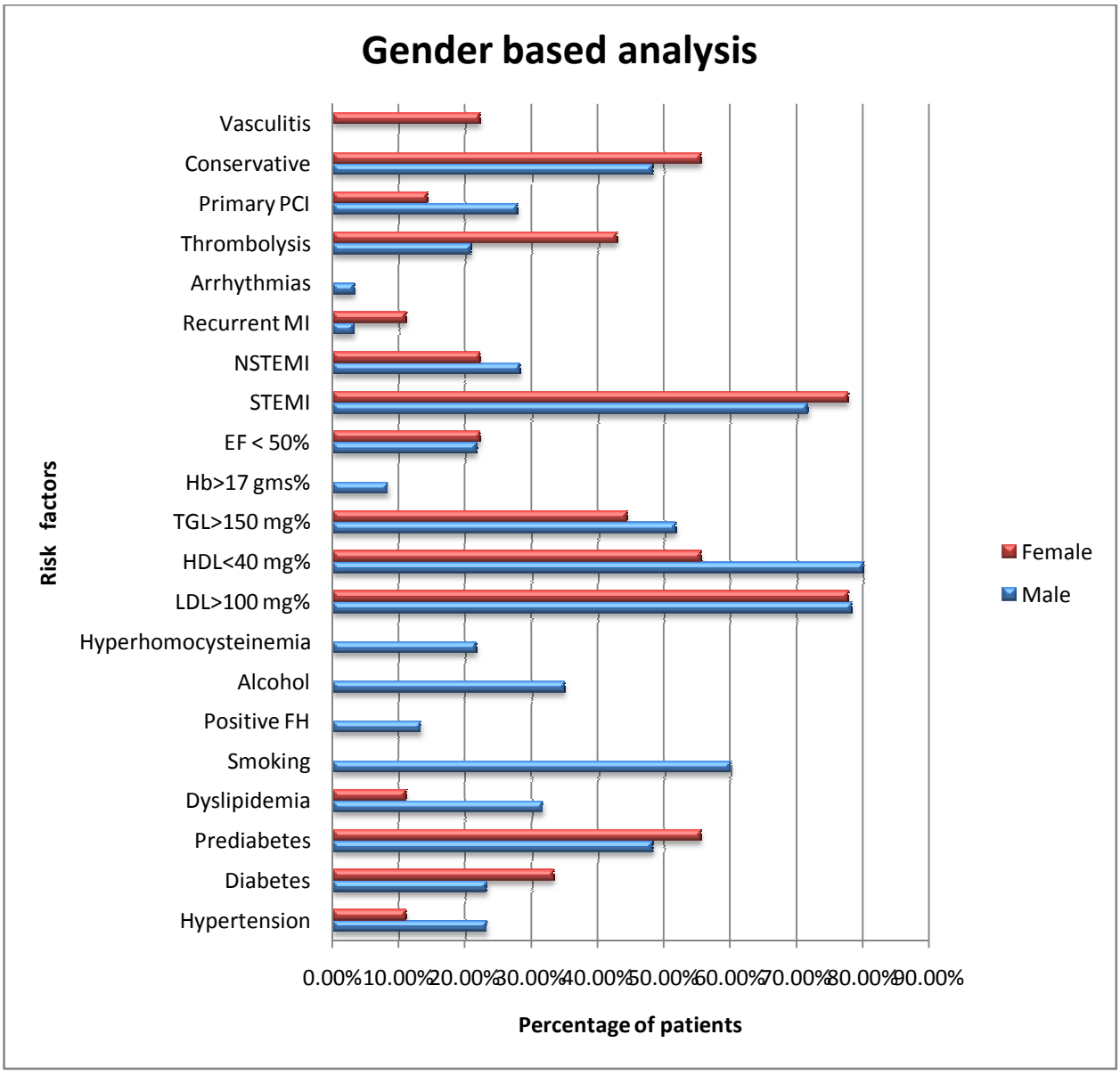
Gender Based Analysis

	MALE	FEMALE
Total	60	9
Hypertension	**14[23.3%]**	1[11.1%]
Diabetes	14[23.3 %]	**3[33.3 %]**
Prediabetes	**29[48.3 %]**	**5[55.6 %]**
Dyslipidemia	19[31.7 %]	1[11.1 %]
smoking	**36[60 %]**	0
Positive FH	8[13.3 %]	0
Alcohol	21[35.0 %]	0
hyperhomocysteinemia	13[21.7 %]	0
LDL > 100 mg %	47[78.3 %]	7[77.8 %]
HDL < 40 mg %	**48[80 %]**	5[55.6 %]
TGL >150 mg %	31[51.7 %]	4[44.4 %]
Hb > 17 gms %	5[8.3 %]	0
EF < 50 %	13 [21.7 %]	2[22.2 %]
STEMI	43[71.7 %]	7[77.8 %]
NSTEMI	17[28.3 %]	2[22.2 %]
RECURRENT MI	2[3.3 %]	1[11.1 %]
Arrhythmias	2[3.3 %]	0
Thrombolysis	9[20.9 %]	3[42.85 %]
Primary PCI	**12[27.9 %]**	1[14.3 %]
Conservative	22[51.2 %]	3[42.85 %]
Vasculitis	0	**2[22.2 %]**

Gender based analysis showed

1. Higher incidence of hypertension among male patients(23.3 % vs 11.1 %)
2. Higher incidence of diabetes among female patients (33.3 % vs 23.3 %)
3. Higher incidence of low HDL among male patients (80 % vs 55.6 %)
4. Higher incidence of prediabetes among both sexes, 48.3 % and 55.6 % in males, females respectively.

5. Incidence of smoking among males was 60 % whereas none of the female patients were smoking
6. Male patients underwent primary PCI more frequently than female patients(27.9 % vs 14.3 %)
7. Vasculitis was presents in 22.2 % of females and none of the males.



DISCUSSION

DISCUSSION

There are many studies of premature CAD conducted in different countries which looked into different aspects of the disease like genetic factors, etiological factors, angiographic findings and outcomes. The most consistent findings in all these studies are the predominance of male sex, higher incidence of smoking and dyslipidemia.

In our study, patients up to 40 years of age were selected, according to the criterion in the literature, which considers a patient with acute myocardial infarction young if he or she is 40 or 45 years old[1-5]. The similarity of age between females and males allowed an adequate comparison between the sexes.

Sex seems to influence the clinical presentation of acute myocardial infarction. Females with acute myocardial infarction are 10 years older than males. In our study, males outnumbered females by more than six times (60 vs 9). Male predominance has been noted in all similar studies in literature.

In a study conducted in Brazil[147], which specifically compared premature CAD in male, female patients, no significant difference was observed between the sexes in risk factors, pattern of coronary artery obstruction and left ventricular function. Female sex and diabetes were independent factors related to the occurrence of reinfarction and death..

When individual risk factors were compared among male and female patients of our study, male patients had a higher prevalence of hypertension (23.3 % versus 11.1 %) whereas female patients had higher prevalence of diabetes (33.3 % versus 23.3 %). Prediabetes prevalence was high in both groups (48.3 % & 56.6 %). Prevalence of high LDL (78.3 % & 77.8 %) and hypertriglyceridemia (51.7 % & 44.4 %) are comparable between sexes but prevalence of low HDL is significantly high among males (80 % & 55.6 %). Similar result for low HDL has been observed in the Brazilian study[147].

Among our patients, only male patients had history of smoking (60 %) and alcoholism (35 %). Hyperhomocysteinemia was present only among male patients, the incidence of which was 21.75 % in our study.

Vasculitis was present only among female patients, the incidence of which is 22.2 %. Vasculitis is an important cause of MI in patients with normal coronaries which needs to be looked for[148].

Haemoglobin level of more than 17 grams % was noted only among male patients (8.3 %) which can be explained by the high incidence (60 %) of smoking only among males in our study. Age and sex related increases in iron stores have been linked to the pathogenesis of several common diseases, including atherosclerosis. Interest in this hypothesis is stimulated by its capacity to explain the sex difference in atherosclerotic diseases and the option of preventive lowering of iron stores by repeated phlebotomy. Iron catalyzes the formation of reactive oxygen species through the Fenton and Haber–Weiss reactions. Free radicals cause lipid peroxidation, leading to the modification of LDL at the molecular level, facilitating its deposition and leading to the formation of atherosclerotic plaque.[149]

Divergent information is available on the relationship between body iron stores and CAD. It has been shown that the concentrations of body iron stores are a strong predictor of CAD in eastern Finnish men.[150] Routine voluntary blood donation is associated, epidemiologically, with reduced coronary risk.[151-2] Furthermore, Kiechl et al[153] found a highly significant correlation between serum ferritin concentration and pathologic carotid artery wall thickening in a longitudinal cohort study. However, several epidemiologic studies investigating the association between high body iron stores and risk of cardiovascular disease in humans have not provided positive results.[154-5] The association between ferritin and CAD was more pronounced in male patients <50 years. Ferritin was significantly higher in diabetic

male patients in comparison with nondiabetic male patients. No association was observed between ferritin and CAD among female patients.[156]

Mode of presentation was comparable in both males and females. They are more likely to present as STEMI (71.7 % & 77.8 %) than as NSTEMI (28.3 % & 22.2 %)

When treatment was analyzed, male patients underwent primary PCI more frequently than female patients (27.9 % versus 14.3 %). In the Brazilian study, the time interval between symptom onset and treatment was longer in females, who underwent thrombolysis and angioplasty less frequently than males did, but not myocardial revascularisation.

Previous similar studies in the literature have shown a lower incidence of hypertension and diabetes mellitus in young patients presenting with myocardial infarction compared with older patients with MI. In our study the prevalence of hypertension was 21.7 %, whereas the prevalence of diabetes mellitus was 24.6 %. Hypertension was more prevalent among males (23.3 % vs 11.1 %) and diabetes was more prevalent among females (33.3 % vs 11.1 %).

The incidence of prediabetes was 49.27 % in our study, which is a new and alarming finding. Its prevalence is high irrespective of sex, income and urban or rural origin. This correlates with the rising incidence of sedentary lifestyle, lack of physical activity and unhealthy food habits among Indians. The higher prevalence of prediabetes among patients with MI at young age indicates that endothelial dysfunction is more likely to occur in the early stages of diabetes or prediabetes which predispose to myocardial infarction. Hence, it becomes necessary to identify and target these patients with primary prevention.

In the Chennai based CUPS STUDY, The overall prevalence rate of CAD is 11.0%. The prevalence rates of CAD were 9.1%, 14.9% and 21.4% in those with normal glucose tolerance (NGT), impaired glucose tolerance (IGT) and diabetes, respectively.

In a meta analysis of studies of prediabetes[133], 18 publications were analysed. Impaired fasting glucose and IGT are associated with modest increases in the risk for cardiovascular disease.[157] Diabetes and CAD occur together more commonly than usually recognized, with the negative impact of dysglycemia apparent before diabetes. In the Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Europe (DECODE), a group of studies of 25,000 individuals were analyzed together. It showed that 2 hr glucose levels increased with age, with less evidence for such an increase in fasting glucose and 2 hr glucose predicted all-cause mortality and CVD risk better than fasting glucose[134]. There was a continuous increase in CVD risk with increasing 2 hr glucose, a phenomenon not clearly found for fasting glucose. Adjusting for fasting glucose, 2 hr glucose continued to significantly predict increased CVD, coronary heart disease (CHD), and total mortality. Furthermore, although the absolute risk was greater for diabetic individuals, in DECODE there were more excess deaths related to hyperglycemia among men with IGT than among those with diabetes because of the greater prevalence of the former group, implying that this group could benefit from treatment. Diabetes Epidemiology: Collaborative analysis Of Diagnostic criteria in Asia (DECODA), a similar study, showed similar data, with the effect of fasting glucose on risk eliminated by adjusting for 2 hr glucose, whereas adjustment for fasting glucose failed to eliminate the association of 2 hr glucose with CVD, CHD, stroke, and total mortality[158]

European Society of Cardiology recommendation on Prediabetes and CAD

Recommendation	Class	Level of evidence
Relationship between hyperglycaemia and CVD is a continuum. For each 1% increase of HbA1c there is a defined increased risk for CVD	I A	A
Risk of CVD for people with overt diabetes is increased by 2-3 times for men and 3-5 times for women when compared with people without diabetes	I A	A
Post-prandial glucose predicts future risk for CVD better than fasting glucose, and elevated post-prandial glucose also predicts increased CV risk in subjects with normal fasting glucose levels	I A	A
Improved control of post-prandial glycaemia may lower CV risk and mortality	IIb C	C
Glucometabolic perturbations carry a particularly high risk for CV morbidity and mortality in women, who in	IIa B	B
People with diabetes and IGT have an increased risk for stroke	I A	A
In stroke patients, unrecognized hyperglycaemia is mostly high post-load glucose seen in the OGTT, whereas the measurement of fasting glucose is insensitive in detecting unrecognized hyperglycemia	I B	B

The prevalence of abnormal lipid profile is very high among the patients in our study which was the finding in similar studies in literature. Our patients had higher prevalence of high LDL (78.26 %) and low HDL(76.82 %) compared to high triglyceride levels (50.72 %). Incidence of low HDL was higher among males than females (80 % vs 55.6 %)

In the CUPS (Chennai Urban Population Study)[159], Prevalence of CAD increased with an increase in total cholesterol, LDL cholesterol, triglycerides and total cholesterol/high-density lipoprotein ratio. Multiple logistic regression analysis identified age and LDL cholesterol as the risk factors for CAD. It has been reported in the literature that the isolated value of LDL-cholesterol plays a less significant role as a cardiovascular risk factor in females as compared with that in males[160-62]. On the other hand, a low HDL-cholesterol level has been considered an important predictor of mortality among females. A meta-analysis carried out at the National Heart, Lung, and Blood Institute has shown that total Hypercholesterolemia and high LDL cholesterol levels correlate with a higher cardiovascular mortality in females younger than 65 years old, but not in the elderly 40. The role of triglycerides as an independent risk factor of coronary artery disease is still controversial. Some admit their greater importance in females, especially in the elderly 42-45, mainly due to an increase in thrombotic risk.

Chen[118] (1995) stated that in addition to previously identified risk factors such as family history and smoking, high plasma triglyceride and low HDL cholesterol levels are associated with premature coronary artery disease.

Tewari [119](2005) observed significant differences in the clinical, biochemical and angiographic profile of young patients with coronary artery disease as compared to elderly patients in north India. Young patients had more atherogenic lipid profile, higher prevalence of smoking and more frequent single vessel disease. He stated that total cholesterol/ high

density lipoprotein cholesterol ratio was a better predictor of coronary artery disease as compared to individual lipid levels.

Hyperhomocysteinemia (>15 mcmol %)was present in 13 patients(18.8 %), only among male patients, the significance of which is difficult to assess, given the small number of patients studied.

Vasculitis was present in 2 of 9 female patients (22.2 %) and none of the male patients. Davies [120](2007) highlights the diagnosis of antiphospholipid syndrome (APS) in five young patients presenting with myocardial infarction and normal coronary arteries at angiography. He states that the diagnosis of antiphospholipid antibody syndrome makes a crucial decision in treatment as they do not require anti atherosclerotic treatment but they do well on high dose warfarin.

Majority of the young MI patients had good left ventricular systolic function with ejection fraction more than 50 % (78.25 %). 18. 95 % of patients had mild LV dysfunction and 2.9 % of patients had moderate LV dysfunction. None of the patients had cardiogenic shock.

Among the patients presented with STEMI, 70 % of patients underwent revascularisation either with thrombolysis (44 %) or primary PCI (26 %). Remaining 30 % were managed conservatively. The most common reason for this is late presentation.

The most common infarct related artery in our study is left anterior descending artery (47.8 %) followed by right coronary artery in 8.7 % of patients. Left circumflex artery is least commonly involved (5.8 %). Younger patients have a higher incidence of normal coronary arteries, mild luminal irregularities, and single vessel coronary artery disease than do older patients.

One of the largest reports of angiographic findings in young patients with CHD comes from a sub study of the CASS trial[163], which compared the results of coronary angiography in 504 young men (≤ 35 years of age) and women (≤ 45 years of age) with a history of an MI to those in over 8300 older patients. The following significant differences were noted:

- Normal coronary arteries were more common in the young patients (18 versus 3 percent). Young women had a higher frequency of angiographically normal coronary arteries than young men, despite a 10 year age difference in the definition of "young."
- Single vessel coronary disease was more common (38 versus 24 percent) and three vessel disease was less common (14 versus 39 percent) in the younger patients.
- Although some series[165-6] have shown a predilection for involvement of the left anterior descending artery in young patients, this was not found in the CASS sub study.
- In another large series of 823 young patients with CHD[169], single vessel disease was present in 55 to 60 percent.

Spontaneous coronary artery dissection is a rare cause of acute MI that is more common in younger patients (under age 50) and in women. In women, the risk of spontaneous coronary dissection appears to be increased during the peripartum period.

Kawasaki disease is one of the most important cause of coronary vasculitis, leading to coronary aneurysm formation in 20 to 25 percent of untreated patients during the acute stage of the disease. Nearly half of acute aneurysms regress, but approximately 20 percent lead to the development of coronary stenosis in the long term. Patients can present with MI or sudden cardiac death (SCD).

The prevalence of significant coronary disease is lower in women presenting with chest pain than in men [168-9]. This was illustrated in a report of 886 patients referred for angiographic evaluation of presumed angina, 23 percent of whom were women[168]. Normal coronary arteries were much more common in women (41 versus 8 percent in men). Myocardial ischemia is present in a minority of the women with normal coronary arteries (20 percent in two reports), perhaps due to microvascular disease[170-1].

Pineda[121] (2008) conducted a retrospective case control study of premature coronary disease with consecutive older patients as controls. It showed that premature CAD is a more frequent entity and it affects predominantly the male sex and shows high prevalence of cardiovascular risk factors, mainly tobacco, dyslipidemia and family history of ischemic heart disease with higher prevalence of single vessel disease and lower initial morbidity.

Comparison with other similar studies

	Our study	J.Pineda et al	Lijia chen et al	Ranjith et al	Hosseini et al	Fournier et al
Total number	69	200	100 males	245	108	108
Age	<40 yrs	< 45 yrs	<40 yrs		<35 yrs	<40 yrs
Males	86.95 %	92.5 %	100		92.6 %	94.5 %
Hypertension	21.7 %	28.5 %	25%	21.63 %	13.9 %	24 %
Diabetes	24.6 %	11.5 %	5%	19.2 %	10. 3 %	12 %
Prediabetes	49.27 %					
Smoking	53.6 %	86.1 %	73 %	74.3 %	39.8 %	94.5 %
Family History	11.6 %	41.5 %	39 %		34.6 %	
Alcohol	30.4 %					22 %
Obesity	7.2 %					37 %
LDL > 100 mg/dl	78.26 %	73.5 %			47.2 %	48 %
HDL < 40 mg/ dl	76.82 %					
TGL > 150 mg/dl	50.72 %					
Hb >17 gm %	7.25 %					
STEMI	72.5 %					
LV EF < 50 %	21.75 %					40 %

Comparison of Angiographic findings

	Our study	Fournier et al	Lijia et al	Hosseini et al
Total number	69	87	100	108
Single vessel	43(62.32 %)	43(49 %)	54	37 (34.3 %)
Double vessel	7(10.15 %)	18(21 %)	36	38 (35.2 %)
Triple vessel	7(10.15 %)	7(8 %)	10	
LAD	33(47.8 %)	32(36.8 %)	37 %	60 (55.6 %)
RCA	6(8.7 %)	40(46 %)	36 %	42 (38.9 %)
LCX	4(5.8 %)	20(23 %)	27 %	27 (25 %)
Recanalised	12(17.4 %)	17(20 %)		18.5 %
Thrombus	26(37.68 %)			

Limitations of the study

- This is single centre study.
- Number of patients studied is small.
- Duration of study is short; hence follow up of patients was not included in the study.
- Large prospective studies with longer follow up are required for better understanding and management of premature CAD.

CONCLUSION

CONCLUSION

Although myocardial infarction, fortunately, is an uncommon entity in young adults aged less than 40 years, it constitutes an important problem for both the patient and the treating physician. They also have a different risk factor profile, clinical presentation, and prognosis in comparison with older patients, which has to be taken into consideration when treating them. The increasing prevalence of risk factors for CAD among young individuals may set up an alarming trend.

In our study, male sex, dyslipidemia and smoking are the most important risk factors for premature CAD which correlates with the results of previous similar studies. Next to them, prediabetes is alarmingly high in our study patients with a prevalence of 49.27 %. Its prevalence is high irrespective of sex, income and rural or urban origin. This is a new observation and indicates the growing trend of unhealthy lifestyle and food habits among young Indian population. Hb A1c needs to be checked in all patients so that early treatment could be initiated even before frank diabetes sets in.

Smoking was more prevalent among rural patients (85.7 %) compared to urban patients (39.6 %) in our study. No significant difference in risk factor profile, presentation was observed among patients with varying income.

Acute STEMI is the most common presentation (72.5 %) in our study and Left ventricular dysfunction is less common (21.75 %). Angiographically, single vessel disease is the most common presentation (62.32 %) with the most common vessel involved being left anterior descending artery (47.8 %). Male patients, urban residents and high income patients are more likely to undergo primary PCI.

Early stabilisation should be followed by risk stratification, and early revascularisation, where appropriate, should be offered as it carries a better clinical outcome.

Risk factors modification, regular physical activity and healthy diet should be emphasised among young patients for primary prevention of premature CAD.

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APPENDIX

APPENDIX I

ABBREVIATIONS

AHA	-	American Heart Association
AMI	-	Acute myocardial infarction
CAD	-	Coronary artery disease
STEMI	-	ST elevation myocardial infarction
CVD	-	Cardio vascular disease
MI	-	Myocardial infarction
NHLBI	-	National Heart, Lung, and Blood Institute
LVF	-	Left ventricular failure
GISSI	-	Italian Group for the Study of Streptokinase in Myocardial Infarction
ISIS	-	Second International Study of Infarct Survival
SAVE	-	Survival and Ventricular Enlargement
ACEI	-	Angiotensin converting enzyme inhibitors
LVAD	-	Left ventricular assist devices
SERCA	-	Sarcoplasmic reticulum Ca2+ ATPase
UA	-	Unstable angina
NSTEMI	-	Non ST segment elevation myocardial infarction
LMWH	-	Low-molecular-weight heparin
LDL	-	Low-density lipoprotein
RNA	-	Ribo nucleic acid
HMG Co A	-	3-Hydroxy-3-methylglutaryl coenzyme A
HDL	-	High-density lipoprotein
CYP	-	Cytochrome P
WHO	-	World Health Organisation

IHD	-	Ischeimic heart disease
HIV	-	Human immunodeficiency virus
AIDS	-	Acquired immune deficiency syndrome
ARB	-	Angiotensin Receptor Blocker
APO	-	Apolipoprotein
PROCAM	-	Prospective Cardiovascular Munster Heart Study
PAR	-	Population attributable risk
OR	-	Odds ratio
CHD	-	Coronary heart disease
ACS	-	Acute coronary syndrome
WC	-	Waist circumference
AGE	-	Advanced glycation end products
ICAM	-	Intracellular adhesion molecules
IL	-	Interleukin
MCP	-	Monocyte chemo attractant protein
NF	-	Nuclear factor
RAGE	-	Receptor for advanced glycation end products
VCAM	-	Vascular cell adhesion molecules
PAI	-	Plasminogen activator inhibitor
RR	-	Relative risk
IGT	-	Impaired glucose tolerance
ADA	-	American diabetic association
NCEP	-	National Cholesterol Education Program
SAM NECP	-	South Asian Modified National Cholesterol Education Program
BMI	-	Body mass index

MS	-	Metabolic syndrome
UKADS	-	United Kingdom Asian Diabetes Study
IDF	-	International Diabetic Federation
LV	-	Left ventricle
ICU	-	Intensive care unit
ECG	-	Electrocardiogram
Hb A1c	-	Hemoglobin A1c
BP	-	Blood pressure
2D	-	Two dimentional
PCI	-	Percutaneous coronary intervention
NGT	-	Normal glucose tolerance
DECODE	-	Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Europe
OGTT	-	Oral Glucose Tolerance Test
CUPS	-	Chennai urban population study
APS	-	Antiphospholipid syndrome
CASS	-	Coronary artery surgery study
KD	-	Kawasaki disease
SCD	-	Sudden cardiac death

APPENDIX II

PROFORMA

Name : Age :

OP No : DOA : DOD :

Residence : Corporation ☐ Municipality ☐ Village ☐

Income < 72,000 ☐ 72,000- 2 lakhs ☐ 2-4 lakhs ☐ > 4 lakhs ☐

Married ☐

Risk Factors

Hypertension ☐ Diabetes ☐ Insulin requiring ☐ Dyslipidemia ☐

Smoking ☐ Family h/o CAD ☐ Vasculitis ☐ AF ☐

Alcohol ☐ BMI ☐ Hyperhomocysteinemia ☐

Investigations

Hb A1c < 5.5 ☐ 5.5 - 6.4 ☐ > 6.5 ☐

LDL < 100 ☐ 100-130 ☐ 130-160 ☐ > 160 ☐

HDL <20 ☐ 21-39 ☐ 40- 49 ☐ 50-59 ☐ > 60 ☐

TGL <150 ☐ 151-199 ☐ 200-249 ☐ >250 ☐

Hb < 10 ☐ 10.1- 13 ☐ 13.1-15.0 ☐ 15.1- 17.0 ☐ > 17 ☐

MCV Normal ☐ Low ☐ High ☐

Clinical Presentation

ACS ☐ STEMI ☐ NSTEMI/USA ☐ Chronic IHD ☐ table Angina ☐

Ischemic HF ☐ Silent IHD ☐

Echo EF >50% ☐ 40-50 % ☐ 30-40% ☐ <30% ☐

In hospital complications

Death ☐ Recurrent MI ☐ CVA ☐ Mechanical ☐ ☐ MR/VSR

Arrhythmias ☐ Pericardial effusion ☐

Treatment Thrombolysis ☐ Primary PCI ☐ Conservative ☐

Angiographic findings

SVD ☐ DVD ☐ TVD ☐

LAD + Diagonal ☐ Lt Cx ☐ RCA ☐

Reanalyzed ☐ Type A ☐ Type B1 ☐ Type B2 ☐ Type C ☐

Thrombus ☐

MASTER CHART

							Risk factors									
S.No	Name	Age	Gender	Residence	Income	Married	Hypertension	Diabetes	Insulin requiring	Dyslipidemia	Smoking	Family h/o CAD	Vasculitis	AF	Alcohol	BMI
1	Senthilkumar	35	1	1	1	2	2	2	2	1	1	1	2	2	1	2
2	Nizaar Ali	35	1	2	1	2	2	2	2	2	1	2	2	2	2	2
3	Lakshmi kandan	38	1	1	1	2	1	2	2	2	2	2	2	2	2	2
4	Shakthivel S	37	1	3	1	2	2	2	2	2	1	1	2	2	2	2
5	Thangaraj	32	1	2	1	2	2	2	2	1	2	2	2	2	1	2
6	Tajudeen	38	1	3	1	2	2	2	2	2	1	2	2	2	2	2
7	Angusamy	36	1	2	4	2	1	1	2	1	1	2	2	2	2	2
8	Sivasudan	39	1	2	3	2	2	1	2	2	2	2	2	2	2	2
9	Arignar Anna	37	1	2	4	2	2	2	2	1	1	1	2	2	1	2
10	Chinnasamy	37	1	3	2	2	1	2	2	1	1	2	2	2	2	2
11	Vijayakumar	35	1	2	2	2	2	2	2	2	1	2	2	2	1	2
12	Balraj	40	1	3	1	2	2	2	2	2	1	2	2	2	2	2
13	Prabhu S	38	1	2	4	2	2	2	2	2	1	2	2	2	1	2
14	Jegadesan	40	1	3	4	2	2	2	2	1	2	2	2	2	2	2
15	Raja	34	1	1	1	2	2	2	2	2	1	2	2	2	1	2
16	Unnikrishnan	28	1	2	2	2	2	2	2	1	1	2	2	2	1	2
17	Selvaraj	40	1	1	1	2	2	1	2	2	2	2	2	2	2	2
18	Gandhimathi	38	2	1	1	2	2	1	1	1	2	2	2	2	2	2
19	Ganesan	34	1	3	2	2	2	1	2	2	2	2	2	2	2	2
20	Nandhini	38	2	2	1	2	1	1	2	2	2	2	2	2	2	2
21	Karthick	25	1	3	2	1	2	2	2	2	1	2	2	2	2	2
22	Anandhan	40	1	3	1	2	2	2	2	1	1	2	2	2	1	2
23	Gunasekar	35	1	2	3	2	2	1	2	1	1	2	2	2	1	2
24	Nagaraj	35	1	1	1	2	2	2	2	2	2	1	2	2	2	2
25	Sivakumar P	39	1	2	1	2	2	1	2	2	1	2	2	2	2	2
26	Rajendranm	34	1	1	2	2	2	2	2	P	2	1	2	2	1	2
27	Jagadeesh T	26	1	3	1	2	2	2	2	1	1	1	2	2	2	2
28	Babu C	37	1	1	2	2	2	2	2	1	2	2	2	2	1	2
29	Venkatachalam	40	1	2	2	2	1	2	2	2	2	2	2	2	2	2
30	Elumalai	40	1	2	1	2	2	2	2	2	1	2	2	2	1	2
31	Maheshwaran	39	1	3	1	2	2	2	2	2	1	1	2	2	1	2
32	Prakash	29	1	2	2	1	2	2	2	2	2	2	2	2	2	2
33	Kulanthaivel	40	1	3	2	2	2	2	2	1	1	2	2	2	1	2
34	Vijayakumar	37	1	1	2	2	1	1	2	2	1	2	2	2	1	2

S.No	Name	Age	Gender	Residence	Income	Married	Hypertension	Diabetes	Insulin requiring	Dyslipidemia	Smoking	Family h/o CAD	Vasculitis	AF	Alcohol	BMI
35	Chinnasamy	38	1	3	1	2	1	2	2	2	1	2	2	2	2	2
36	Mahendran	38	1	3	4	2	1	2	2	2	1	2	2	2	2	2
37	Zaltihar Ahmed Basha	38	1	1	2	2	1	2	2	2	2	2	2	2	2	2
38	Subramaniam	35	1	3	1	2	2	2	2	2	1	2	2	2	2	2
39	Selvaraj	35	1	3	2	1	1	1	2	2	1	2	2	2	2	1
40	Anandhan B	40	1	1	1	2	2	2	2	2	2	2	2	2	2	2
41	Kumarasamy	32	1	2	1	2	2	1	2	2	2	2	2	2	2	2
42	Manikandan	40	1	1	1	2	2	1	2	2	2	2	2	2	2	1
43	Geetha	40	2	1	1	2	2	1	1	2	2	2	2	2	2	2
44	Sarojini	40	2	1	1	2	2	2	2	2	2	2	2	2	2	2
45	Vimalathithan	37	1	3	4	2	1	2	2	2	1	2	2	2	2	1
46	Velusamy	40	1	2	4	2	2	2	2	2	2	2	2	2	2	2
47	Murugesan	33	1	2	2	2	2	2	2	1	1	2	2	2	2	2
48	Saravannan	38	1	3	2	2	1	2	2	2	1	2	2	2	2	2
49	Srinivasan V	36	1	3	1	2	2	2	2	2	2	2	2	2	1	2
50	Hemalatha.S	22	2	1	3	1	2	2	2	2	2	2	1	2	2	2
51	Robert Kennedy	37	1	1	2	2	2	1	2	1	2	2	2	2	2	2
52	Saradha	33	1	2	1	2	2	2	2	2	2	2	2	2	2	2
53	Saravanan G	38	1	2	2	1	1	2	2	2	2	2	2	2	2	2
54	Subramanian	40	1	3	2	2	2	2	2	2	1	2	2	2	2	2
55	Senthilkumar A	33	1	1	1	2	2	2	2	2	2	2	2	2	2	1
56	Pandiyan	35	1	1	1	2	2	2	2	1	1	1	2	2	2	2
57	Palanisamy A	32	1	1	2	2	2	2	2	1	1	2	2	2	1	2
58	Shanmugasundaram	39	1	1	3	2	2	1	2	1	2	2	2	2	1	2
59	Ratna	40	1	1	1	2	2	2	2	2	2	2	2	2	2	2
60	George Saman	30	1	1	1	2	2	2	2	2	1	2	2	2	2	2
61	Mohanasundaram	35	1	1	2	2	2	1	2	2	2	2	2	2	2	1
62	Mohan C	38	1	3	2	2	2	1	2	1	1	2	2	2	1	2
63	Marimuthu	37	1	2	2	1	2	2	2	2	1	2	2	2	2	2
64	Gurusamy	32	1	1	1	2	1	2	2	2	2	2	2	2	1	2
65	Marimuthu	37	1	2	2	2	2	2	2	2	2	2	2	2	2	2
66	Saira Moosa	36	2	1	3	2	2	2	2	2	2	2	1	2	2	2
67	Ragupathy	37	1	1	1	2	2	2	2	1	1	2	2	2	1	2
68	Palanisamy	40	1	1	3	2	1	2	2	2	1	2	2	2	2	2
69	Rejendran K	40	1	3	1	2	2	2	2	2	1	2	2	2	2	2

			Investigations						Clinical Presentation								In Hospital Complications			
S.No	Name	Hyperhomo cystenemia	HbA1c	LDL	HDL	TGL	Hb	MCV	ACS	STEMI	NSTEMI	Chronic IHD	Stable Angina	Ischemic HF	Silent IHD	Echo EF	Death	Recurrent MI	CVA	Mechanical
1	Senthilkumar	1	2	2	2	3	5	3	2	1	2	2	2	2	2	1	2	2	2	2
2	Nizaar Ali	2	2	1	2	1	3	1	2	1	1	2	2	2	2	1	2	2	2	2
3	Lakshmi kandan	2	1	2	1	1	4	1	2	1	2	2	2	2	2	1	2	2	2	2
4	Shakthivel S	2	1	2	2	3	3	1	2	1	2	2	2	2	2	1	2	2	2	2
5	Thangaraj	1	1	1	2	1	2	2	2	1	2	2	2	2	2	1	2	2	2	2
6	Tajudeen	2	2	3	3	1	3	1	2	1	2	2	2	2	2	1	2	2	2	2
7	Angusamy	2	3	4	2	1	3	1	2	1	2	2	2	2	2	1	2	2	2	2
8	Sivasudan	2	1	4	3	4	3	1	2	2	2	2	2	2	2	1	2	2	2	2
9	Arignar Anna	2	3	3	2	4	4	1	2	1	2	2	2	2	2	1	2	2	2	2
10	Chinnasamy	2	1	2	2	2	4	1	2	2	1	2	2	2	2	1	2	2	2	2
11	Vijayakumar	2	1	4	2	4	4	1	2	1	2	2	2	2	2	2	2	2	2	2
12	Balraj	2	2	2	2	1	3	1	2	2	1	2	2	2	2	1	2	2	2	2
13	Prabhu S	2	2	3	2	3	3	1	2	1	2	2	2	2	2	1	2	2	2	2
14	Jegadesan	2	3	4	2	2	3	1	2	1	2	2	2	2	2	3	2	2	2	2
15	Raja	2	1	4	4	1	3	3	2	1	2	2	2	2	2	2	2	2	2	2
16	Unnikrishnan	2	2	3	2	3	2		2	1	2	2	2	2	2	1	2	2	2	2
17	Selvaraj	2	1	4	2	2	4	1	2	2	2	2	1	2	2	1	2	2	2	2
18	Gandhimathi	2	3	4	3	2	3	1	2	1	2	2	2	2	2	1	2	2	2	2
19	Ganesan	2	1	2	4	1	3	1	2	1	2	2	2	2	2	1	2	2	2	2
20	Nandhini	2	3	3	2	2	3	1	1	2	2	2	2	1	2	1	2	2	2	2
21	Karthick	2	1	3	3	1	4	1	2	1	2	2	2	2	2	1	2	2	2	2
22	Anandhan	1	2	2	4	1	3	1	2	1	2	2	2	2	2	2	2	2	2	2
23	Gunasekar	2	3	4	2	4	4	3	2	1	2	2	2	2	2	1	2	2	2	2
24	Nagaraj	1	1	3	2	1	3	3	2	1	2	2	2	2	2	1	2	2	2	2
25	Sivakumar P	2	3	3	2	4	4	3	2	2	1	2	2	2	2	1	2	2	2	2
26	Rajendranm	1	3	1	2	1	3	1	2	1	2	2	2	2	2	1	2	2	2	2
27	Jagadeesh T	2	2	1	2	1	3	1	2	1	2	2	2	2	2	1	2	2	2	2
28	Babu C	2	2	4	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2	2
29	Venkatachalam	2	2	2	2	1	4	1	2	1	2	2	2	2	2	1	2	1	2	2
30	Elumalai	1	1	2	2	1	5	3	2	1	2	2	2	2	2	1	2	2	2	2
31	Maheshwaran	1	2	2	3	4	4	1	2	2	2	2	2	2	2	1	2	2	2	2
32	Prakash	2	2	2	2	1	2	1	2	1	2	2	2	2	2	1	2	2	2	2
33	Kulanthaivel	2	3	4	2	2	4	3	2	2	1	2	2	2	2	1	2	2	2	2
34	Vijayakumar	2	2	4	4	4	4	3	2	1	2	2	2	2	2	2	2	1	2	2

S.No	Name	Hyperhomo cystenemia	HbA1c	LDL	HDL	TGL	Hb	MCV	ACS	STEMI	NSTEMI	Chronic IHD	Stable Angina	Ischemic HF	Silent IHD	Echo EF	Death	Recurrent MI	CVA	Mechanical
35	Chinnasamy	2	2	2	2	1	4	1	2	1	2	2	2	2	2	1	2	2	2	2
36	Mahendran	2	2	1	2	1	5	1	1	2	2	2	2	2	2	1	2	2	2	2
37	Zaltihar Ahmed Bash	2	2	2	2	1	3	1	2	1	2	2	2	2	2	1	2	2	2	2
38	Subramaniam	2	2	2	2	3	4	1	2	1	2	2	2	2	2	1	2	2	2	2
39	Selvaraj	2	2	3	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2
40	Anandhan B	1	2	3	3	2	4	1	2	1	2	2	2	2	2	2	2	2	2	2
41	Kumarasamy	2	2	1	2	1	3	1	2	1	2	2	2	2	2	1	2	2	2	2
42	Manikandan	2	1	3	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2
43	Geetha	2	3	4	2	2	3	1	2	1	2	2	2	2	2	1	2	1	2	2
44	Sarojini	2	2	2	2	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2
45	Vimalathithan	2	2	2	2	1	2	1	2	1	2	2	2	2	2	1	2	2	2	2
46	Velusamy	1	1	4	3	1	4	1	2	1	2	2	2	2	2	1	2	2	2	2
47	Murugesan	2	2	4	2	3	3		2	2	1	2	2	2	2	1	2	2	2	2
48	Saravannan	1	2	3	2	2	4	1	2	2	2	2	2	2	2	1	2	2	2	2
49	Srinivasan V	1	2	1	2	1	3	1	2	1	2	2	2	2	2	2	2	2	2	2
50	Hemalatha.S	2	2	1	3	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2
51	Robert Kennedy	2	2	4	3	1	2	1	2	1	2	2	2	2	2	1	2	2	2	2
52	Saradha	2	2	3	2	1	2	2	2	2	1	2	2	2	2	1	2	2	2	2
53	Saravanan G	2	2	2	2	1	5	1	2	1	2	2	2	2	2	2	2	2	2	2
54	Subramanian	2	1	1	2	1	4	1	2	1	2	2	2	2	2	1	2	2	2	2
55	Senthilkumar A	2	1	3	3	4	3	3	2	1	2	2	2	2	2	1	2	2	2	2
56	Pandiyan	2	1	2	2	4	4		2	2	1	2	2	2	2	3	2	2	2	2
57	Palanisamy A	1	2	2	2	2	3	1	2	1	2	2	2	2	2	1	2	2	2	2
58	Shanmugasundaram	2	3	2	3	1	4	1	2	2	1	2	2	2	2	1	2	2	2	2
59	Ratna	2	2	2	3	1	2	2	2	1	2	2	2	2	2	1	2	2	2	2
60	George Saman	2	1	1	2	1	5	1	2	1	2	2	2	2	2	1	2	2	2	2
61	Mohanasundaram	2	3	1	2	1	3	1	2	2	1	2	2	2	2	1	2	2	2	2
62	Mohan C	2	3	2	2	2	4	3	2	1	2	2	2	2	2	1	2	2	2	2
63	Marimuthu	2	3	4	2	2	3	2	2	1	2	2	2	2	2	2	2	2	2	2
64	Gurusamy	2	2	1	1	4	3	1	2	2	2	2	2	2	2	1	2	2	2	2
65	Marimuthu	2	2	4	2	2	3	1	2	1	2	2	2	2	1	2	2	2	2	2
66	Saira Moosa	2	1	1	2	1	1	1	2	1	2	2	2	2	2	1	2	2	2	2
67	Ragupathy	1	3	2	2	4	4	1	1	2	2	2	2	2	2	1	2	2	2	2
68	Palanisamy	2	2	1	2	2	4	1	2	1	2	2	2	2	2	1	2	2	2	2
69	Rejendran K	2	1	1	2	1	3	3	2	1	2	2	2	2	2	2	2	2	2	2

In Hospital Complications						Angiographic Findings								
S.No	Name	MR/VSR	Arrhythmias	Pericardial Effusion	Treatment	LAD+ Diagonal	LtCx	RCA	Recana lised	Type A	Type B1	Type B2	Type C	Thrombus
1	Senthilkumar	2	2	2	2	1	2	2	2	2	2	2	2	1
2	Nizaar Ali	2	2	2	1	1	2	2	2	2	2	2	2	2
3	Lakshmi kand	2	2	2	1	1	1	2	2	2	2	2	2	2
4	Shakthivel S	2	2	2	1	1	2	2	2	2	2	2	2	2
5	Thangaraj	2	1	2	1	2	2	2	1	2	2	2	2	2
6	Tajudeen	2	2	2	1	1	2	2	2	2	2	2	2	2
7	Angusamy	2	2	2	3	1	1	1	2	2	2	2	2	2
8	Sivasudan	2	2	2	3	1	1	1	2	2	2	2	2	2
9	Arignar Anna	2	2	2	2	2	1	1	2	2	2	2	2	2
10	Chinnasamy	2	2	2	3	2	1	2	2	2	2	2	2	2
11	Vijayakumar	2	2	2	3	1	2	2	2	2	2	2	2	2
12	Balraj	2	2	2	3	1	2	2	2	2	2	2	2	2
13	Prabhu S	2	2	2	2	1	2	1	2	2	2	2	2	2
14	Jegadesan	2	2	2	3	1	1	1	2	2	2	2	2	2
15	Raja	2	2	2	3	1	2	2	2	2	2	2	2	2
16	Unnikrishnan	2	2	2	1	2	2	1	2	2	2	2	2	1
17	Selvaraj	2	2	2	3	2	1	1	2	2	2	2	2	2
18	Gandhimathi	2	2	2	1	1	2	2	2	2	2	2	2	2
19	Ganesan	2	2	2	1	1	1	2	2	2	2	2	2	2
20	Nandhini	2	2	2	3	2	2	2	2	2	2	2	2	2
21	Karthick	2	2	2	3	1	2	2	2	2	2	2	2	2
22	Anandhan	2	2	2	1	1	2	2	2	2	2	2	2	2
23	Gunasekar	2	2	2	2	1	2	2	2	2	2	2	2	2
24	Nagaraj	2	2	2	2	1	2	2	2	2	2	2	2	2
25	Sivakumar P	2	2	2	2	2	1	2	2	2	2	2	2	2
26	Rajendranm	2	2	2	2	1	2	2	2	2	2	2	2	1
27	Jagadeesh T	2	2	2	1	2	2	2	1	2	2	2	2	2
28	Babu C	2	2	2	1	1	2	2	1	2	2	2	2	2
29	Venkatachala	2	2	2	1	1	2	2	2	2	2	2	2	2
30	Elumalai	2	1	2	1	1	2	2	2	2	2	2	2	2
31	Maheshwarai	2	2	2	3	2	2	2	2	2	2	2	2	2
32	Prakash	2	2	2	3	1	2	2	2	2	2	2	2	2
33	Kulanthaivel	2	2	2	3	2	1	2	2	2	2	2	2	2
34	Vijayakumar	2	2	2	1	1	2	2	1	2	2	2	2	1

S.No	Name	MR/VSR	Arrhythmias	Pericardial Effusion	Treatment	LAD+ Diagonal	LtCx	RCA	Recana lised	Type A	Type B1	Type B2	Type C	Thrombus
35	Chinnasamy	2	2	2	3	1	1	2	2	2	2	2	2	2
36	Mahendran	2	2	2	3	1	2	2	2	2	2	2	2	2
37	Zaltihar Ahme	2	2	2	3	1	2	2	2	2	2	2	2	2
38	Subramaniam	2	2	2	3	1	2	2	2	2	2	2	2	2
39	Selvaraj	2	2	2	2	1	2	2	2	2	2	2	2	2
40	Anandhan B	2	2	2	2	1	2	2	2	2	2	2	2	2
41	Kumarasamy	2	2	2	3	1	2	2	2	2	2	2	2	2
42	Manikandan	2	2	2	1	2	2	2	2	2	2	2	2	2
43	Geetha	2	2	2	3	1	2	2	2	2	2	2	2	2
44	Sarojini	2	2	2	3	2	2	1	2	2	2	2	2	2
45	Vimalathithar	2	2	2	1	2	2	2	2	2	2	2	1	2
46	Velusamy	2	2	2	2	1	2	2	2	2	2	2	2	2
47	Murugesan	2	2	2	3	2	2	1	2	2	2	2	2	2
48	Saravannan	2	2	2	3	1	2	2	2	2	2	2	2	2
49	Srinivasan V	2	2	2	3	1	2	2	2	2	2	2	2	2
50	Hemalatha.S	2	2	2	1	2	2	2	2	2	2	2	2	2
51	Robert Kenne	2	2	2	2	2	2	1	2	2	2	2	2	2
52	Saradha	2	2	2	2	1	2	2	2	2	2	2	2	2
53	Saravanan G	2	2	2	1	1	2	2	2	2	2	2	2	2
54	Subramanian	2	2	2	1	1	2	2	2	2	2	2	2	2
55	Senthilkumar	2	2	2	1	1	2	2	2	2	2	2	2	2
56	Pandiyan	2	2	2	1	2	2	2	2	2	2	2	2	2
57	Palanisamy A	2	2	2	2	1	2	2	2	2	2	2	2	2
58	Shanmugasur	2	2	2	3	1	2	1	2	2	2	2	2	2
59	Ratna	2	2	2	3	2	2	2	2	2	2	2	2	2
60	George Sama	2	2	2	3	2	2	2	2	2	2	2	2	2
61	Mohanasunda	2	2	2	3	1	2	2	2	2	2	2	2	2
62	Mohan C	2	2	2	3	1	2	2	2	2	2	2	2	2
63	Marimuthu	2	2	2	1	1	2	2	2	2	2	2	2	2
64	Gurusamy	2	2	2	3	2	2	2	2	2	2	2	2	2
65	Marimuthu	2	2	2	3	1	2	2	2	2	2	2	2	2
66	Saira Moosa	2	2	2	3	2	2	2	1	2	2	2	2	2
67	Ragupathy	2	2	2	3	1	2	2	2	2	2	2	2	2
68	Palanisamy	2	2	2	3	2	2	1	2	2	2	2	2	2
69	Rejendran K	2	2	2	3	1	2	2	2	2	2	2	2	2

ABSTRACT

INTRODUCTION:

Although coronary artery disease (CAD) primarily occurs in patients over the age of 40, younger patients can still be affected. CAD occurs in Asian Indians 5–10 years earlier than in other populations around the world. Traditional risk factors of CAD are prevalent in young patients with acute STEMI but with a different pattern compared to their older counterparts. While the literature from developed countries is abundant in data highlighting various aspects of myocardial infarction in young patients, only a few studies have been published in India.

MATERIALS AND METHODS

We conducted a prospective observational study of young patients (age < 40 yr, both male and female) admitted with acute coronary syndrome (STEMI and NSTEMI) for a period of one year at P S G Hospital. Detailed history of risk factors and demographic profile were recorded. Complete blood count, fasting lipid profile, Hb A1c, Echocardiogram were done. Treatment modality was noted. Coronary angiogram was done and lesions were analyzed.

RESULTS:

A total of 69 patients were studied. 60 Patient were males (86.95 %), 9 patients were females (13.05 %). 26 patients (37.7%) came from corporation, 22 patients (31.9%) came from municipalities and 21 patients (30.4%) came from villages. 32(46.42%), 24(34.8 %), 7(10.1%) patients had low, medium, high income respectively. 17(24.6%) were diabetics, 37 (53.6%) were smokers, 8 (11.6%) had family H/o CAD, 21(30.4%) were consuming alcohol. 34(49.27%) had prediabetes, 54(78.26%) had LDL cholesterol > 100mg%, 53(76.82 %) had HDL< 40mg %, 35 (50.72%) had TGL level > 150%. 5(7.25%) had Hb >17gm%. 13(18.8%)

had hyperhomocysteinemia. 50(72.5%) had STEMI, 19(27.5%) had NSTEMI. 54(78.25%) had normal LV function. Among the 50 patients who had STEMI, 22 underwent thrombolysis (44 %) and 13(26 %) underwent primary PCI. CAG showed single vessel disease in 43(62.32 %), double vessel disease in 7(10.15 %), triple vessel disease in 7(10.15 %), recanalised vessels in 12(17.4 %), thrombus in 26(37.68%). LAD artery is involved most commonly in 33(47.8 %) followed by RCA in 6 (8.7 %), LCX in 4(5.8 %). Urban patients underwent primary PCI more frequently than rural patients (33.3 % vs 7.14 %). Gender based analysis showed higher incidence of hypertension, low HDL among male patients, higher incidence of diabetes among female patients, higher incidence of prediabetes among both sexes. Incidence of smoking among males was 60 % whereas none of the female patients were smoking. Male patients underwent primary PCI more frequently than female patients (27.9 % vs 14.3 %)

CONCLUSION:

Male sex, dyslipidemia and smoking are the most important risk factors for premature CAD. Prediabetes is alarmingly high in our study with a prevalence of 49.27 %. Its prevalence is high irrespective of sex, income and rural or urban origin. Acute STEMI is the most common presentation (72.5 %) and LV dysfunction is less common (21.75 %). Angiographically, single vessel disease is most common (62.32 %) with the most common vessel involved being left anterior descending artery (47.8 %). Male patients, urban residents and high income patients are more likely to undergo primary PCI.

KEY WORDS:

Premature CAD, young MI, Prediabetes and CAD, Dyslipidenia and CAD, Angiographic profile of premature CAD, clinical profile of premature CAD.